Vol. 68, No. 10 Established 1884 October 1951

ARCHIVES OF PEDIATRICS

A MONTHLY DEVOTED TO THE

DISEASES OF INFANTS AND CHILDREN

JOHN FITCH LANDON, M.D., Editor

LEADING ARTICLES IN THIS NUMBER

Acquired Hemolytic Anemia of the Newborn (Erythroblastosis foetalis) Due to O-B Iso-immunization. Report of Case.
Abraham M. Fromin, M.D., and Albert Kohn, M.D. 461

Congenital Megacolon. Report of Case.
Thomas I. Longworth, M.D. 467

Neuroblastoma. Report of Case.
Myron B. Harkavy, M.D. 473

A Pediatrician Views the Trends in Child Psychiatry.
Joanne Smith, M.D. 477

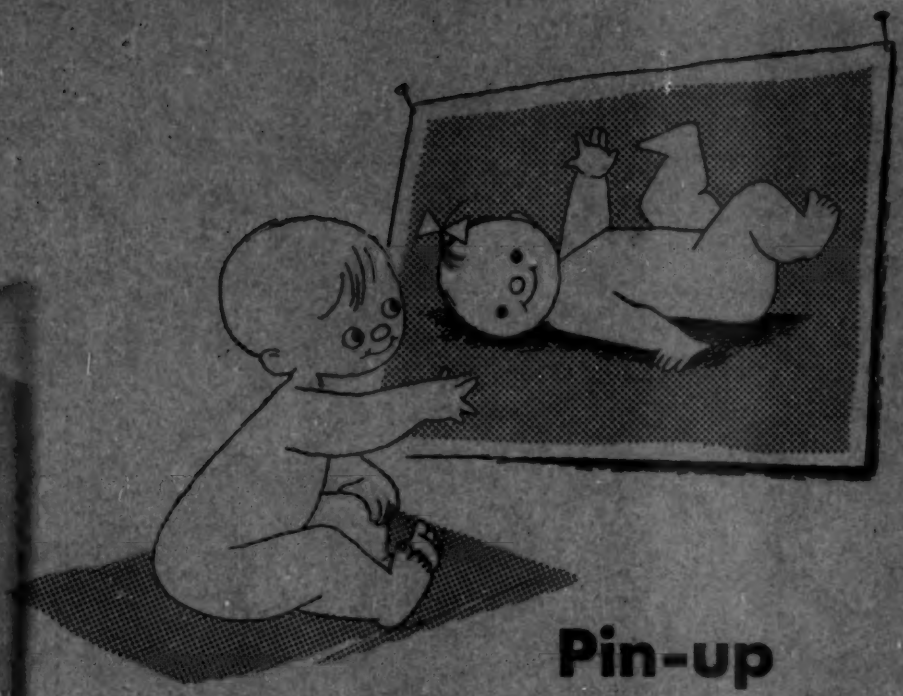
Epidemic Diarrhea of the Newborn.
J. Douglas Hallock, M.D. 488

Pediatrics Half a Century Ago.
Abscess of the Lung Following Acute Pneumonia. Report of Two Cases.
L. Emmett Holt, M.D. 501

E. B. TREAT & CO., Inc., *Publishers*, 45 East 17th Street, NEW YORK, 3
Yearly Subscription $6.00 (Foreign $6.75); Single Copy, $1.00

COPYRIGHT, 1951, BY E. B. TREAT & CO., INC. ALL RIGHTS RESERVED

Entered as second-class matter Feb. 5, 1892, at New York, N. Y., P. O., under the Act of March 3, 1879



Pin-up

SIMILAC-fed infants can "look good" in a deeper sense, too. Closely approximating mother's milk, Similac provides adequate vitamin C supply; the full array of essential amino acids found in human milk; both folic acid and vitamin B_{12}; and a calcium-phosphorus ratio of 1½ to 1—as well as the advantages of zero curd tension.

from birth to birthday . . .

zero curd tension

SIMILAC

all important constituents so modified that there is no closer equivalent to human breast milk

SIMILAC DIVISION • M & R LABORATORIES • *Columbus 16, Ohio*

For their varying vitamin needs

MEAD'S versatile "VI-SOLS"

- water-soluble
- pleasant tasting
- easy to use

Poly-Vi-Sol, Tri-Vi-Sol and Ce-Vi-Sol provide 3 different vitamin combinations to meet particular specifications for individual patients.

Mead's Vi-Sols can be dropped into the mouth or mixed with formula, fruit juice or cereal.

Available in 15 and 50 cc. bottles with calibrated droppers for easy dosage measurement.

	Vitamin A	Vitamin D	Ascorbic Acid	Thiamine	Riboflavin	Niacinamide
POLY-VI-SOL each 0.6 cc. supplies	5000 units	1000 units	50 mg.	1 mg.	0.8 mg.	5 mg.
TRI-VI-SOL each 0.6 cc. supplies	5000 units	1000 units	50 mg.			
CE-VI-SOL each 0.5 cc. supplies			50 mg.			

MEAD'S

MEAD JOHNSON & CO.
EVANSVILLE 21, IND., U.S.A.

Mothers everywhere are praising their doctors for recommending BiB Orange Juice

MRS. LOUISE BELLINE
of Long Island, New York

"No more tiresome preparation, since my doctor recommended Bib. Nothing to squeeze or strain, mix, thaw or boil. And Bib let's me be *sure* my two get their full daily needs of Natural Vitamin C!"

MRS. DAVID WEINBERGER
of Pine Tree Drive, Miami Beach
Mother of one

"It was a happy day for me, when my doctor put Evelyn on Bib. He said Evelyn should have no trouble digesting Bib, because Bib reduces troublesome citrus oils to less than 0.01%! And he was right!"

BIB, a Council-accepted orange juice for babies

You just open . . . pour . . . and feed!

OTHER GOOD REASONS YOUR BABIES WILL DO WELL ON BIB:

Bib specifications accepted by the Council on Foods and Nutrition of the AMA.

CONTENT OF NATURAL VITAMIN C . .	Over 40 mg/100 cc when packed. (1949-50 pack averaged 49 mg/100 cc.)
PEEL OIL CONTENT	Less than 0.01% (Removed by special BIB process—these oils are widely blamed for lack of tolerance to orange juice.)
FLAVOR	Held constant by analysis of Brix-Acid ratio and addition of minimal amounts of dextrose.
BACTERIA COUNT	Under 10/ml. (Fresh juice averages 25,000/ml., frozen averages 15,000/ml.)
PULP CONTENT	BIB reduces fruit cells to microscopic size—retaining natural nutrients while assuring a juice free flowing even through bottle nipples.

Tell your mothers the BiB "6-Second" story

just open . . . pour . . . and feed!

P.S. Drop us a card and we'll send you full-size samples and literature. Write The BIB Corporation, Box 866, Dept. E9, Lakeland, Florida.

THE BIB CORPORATION • LAKELAND, FLORIDA

TO CONTRIBUTORS AND CORRESPONDENTS OF

ARCHIVES OF PEDIATRICS

Subscription $6.00 a Year, in Advance **Foreign $6.75** **Single Copy, $1.00**

Editorial Communications address to JOHN F. LANDON, M.D., 120 East 75th Street, New York 21

Business Communications address to E. B. TREAT & Co., INC., 45 East 17th Street, New York 3

ORIGINAL ARTICLES, brief reports of rare and interesting cases, or new modes of treatment are solicited, but none will be considered for publication except with the distinct understanding that it is contributed exclusively to this journal. Manuscripts must be typewritten, double-spaced, and the original, not the carbon, copy submitted. The editor and publishers will not be responsible for views expressed.

ILLUSTRATIONS, as in the judgment of the editor are necessary, will be furnished free when satisfactory photographs or drawings are supplied. Photographs must be clear and distinct; drawings must be in India ink on white paper.

COPYRIGHT. — Original communications appearing in this journal are protected by copyright and can not be reproduced, either wholly or in part, without permission of the publishers.

REPRINTS of articles appearing among "Original Communications" may be ordered immediately upon receipt of galley proof by communicating direct with the publishers, E. B. Treat & Co., Inc., 45 East 17th Street, New York 3, who will supply their schedule of prices.

DISCONTINUANCES. — The publishers must be notified when a subscriber wishes his journal stopped and all arrearages must be paid. Without such notification it is assumed that a continuance is desired. Journals returned are not notice of discontinuance.

REMITTANCES should be made by check, draft, post office or express money order. If currency is sent, the letter should be registered. Stamps in amounts under one dollar are acceptable.

CHANGE OF ADDRESS NOTICE should give both the old and the new and state if change is permanent or temporary.

CONTENTS

ORIGINAL ARTICLES

Acquired Hemolytic Anemia of the Newborn (Erythroblastosis foetalis) Due to O-B Iso-immunization. Report of Case.
ABRAHAM M. FRUMIN, M.D., AND ALBERT KOHN, M.D. 461

Congenital Megacolon. Report of Case.
THOMAS I. LONGWORTH, M.D. 467

Neuroblastoma. Report of Case.
MYRON B. HARKAVY, M.D. 473

A Pediatrician Views the Trends in Child Pyschiatry.
JEANNE SMITH, M.D. 477

(Continued on page 5)

Case report*—Jimmy and Johnny E., twins. Birth one month premature—"shadowed" by placenta previa and delivery by section. The twins are S-M-A babies from birth. The mother's condition was so poor that her own survival was in doubt. Therefore, there was little opportunity to do more than prepare the S-M-A formula. The babies received no supplementary vitamins. Weight at 8 months of age, 22 pounds (one underwent surgery for hernia at the age of 6 weeks).

**Volunteered by the proud mother, supported by data supplied by attending physician (name on request).*

S-M-A®

with Vitamin C added

builds husky babies

Supplied:
S-M-A Concentrated Liquid—cans of 13 fl. oz.
S-M-A Powder—cans of 1 lb.

Wyeth®

Wyeth INCORPORATED • PHILADELPHIA 2, PA.

(Continued from page 3)

CLINICAL REVIEW

Epidemic Diarrhea of the Newborn.
J. Douglas Hallock, M.D. 488

PEDIATRICS HALF A CENTURY AGO

Abscess of the Lung Following Acute Pneumonia. Report of Two Cases.
L. Emmett Holt, M.D. 501

DEPARTMENT OF ABSTRACTS

Lans, H. S., et al.: Potassium Deficiency in Bulbar Poliomyelitis..... 509

Thwaite, W. G.: Bronchiogenic Carcinoma in a Twelve-Year-Old Boy..... 509

Schachter, M., and Cotte, S.: A Study of the Importance of the Sex Factor in Early Development..... 510

ITEMS

Treatment of Yaws by Aureomycin..... 472

Heredity of Diabetes Mellitus..... 487

Prefrontal Leukotomy in Minors..... 500

Role of Adrenal in Pink Disease..... 508

Tuberculous Meningitis Cured by Streptomycin..... 508

in whooping cough

ELIXIR BROMAURATE

GIVES EXCELLENT RESULTS

Cuts short the period of illness and relieves the distressing spasmodic cough. Also valuable in **Bronchitis** and **Bronchial Asthma.**

In four-ounce original bottles. A teaspoonful every 3 to 4 hours.

Extensively Used in Pediatric Practice.

GOLD PHARMACAL CO. NEW YORK CITY

WHEN *mild sedation* IS NEEDED

TRY THIS *NEW* VEHICLE

IT'S the improved, better-tasting NEMBUTAL Elixir—a pleasant change of sedative for patients who periodically require sedation and for whom phenobarbital or bromides have been previously prescribed.

Consider these advantages: attractive orange color, pleasant odor and much better taste than the old Elixir. Onset of action is prompt and duration may be short or prolonged, depending on the dosage. As short-acting NEMBUTAL is quickly and completely destroyed in the body, there is usually no "hangover" and little tendency toward cumulative effect.

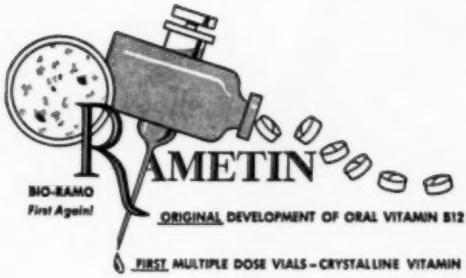
One teaspoonful (1 fl.dr.) of the Elixir represents 15 mg. (¼ gr.) of NEMBUTAL Sodium, making it easy to administer small doses for mild effect.

Miscibility of the new Elixir is greater than that of the old, and compatibility is much wider. Taste and miscibility were improved by the substitution of SUCARYL® (Cyclamate, Abbott), a heat-stable, non-caloric sweetener, for much of the sugar.

Short-acting NEMBUTAL affords any desired degree of cerebral depression—from mild sedation to deep hypnosis. In the complete NEMBUTAL line are capsules, tablets, suppositories, elixirs, solutions and sterile powder for solutions. Small-dosage sizes simplify oral, rectal or intravenous administration. Abbott

TRY THE IMPROVED, BETTER-TASTING **Nembutal® Elixir**

(PENTOBARBITAL, ABBOTT)



For Economical Vitamin B_{12} Therapy

RAMETIN (crystalline Vitamin B_{12} U.S.P.) has been widely accepted by the medical profession and contains the ONLY vitamin B_{12} official in the U.S.P., XIV.

RAMETIN TABLETS—the **first oral** vitamin B_{12}. Palatable, candy-like in taste, soluble scored tablets. Available in three potencies:

5 microgram tablet-bottle of 25 and 100

10 microgram tablet-bottle of 100.

30 microgram tablet-bottle of 100.

RAMETIN INJECTION—A.M.A. COUNCIL ACCEPTED. the **first multiple dose vial** to be offered to the medical profession and Council Accepted. Available in:

10 cc. vials—10 micrograms and 15 micrograms per cc. of Crystalline Vitamin B_{12} U.S.P., XIV.

LITERATURE ON REQUEST

BIO-RAMO DRUG CO., INC.
Baltimore 1, Md.

AS THEY FIND IT—"PRACTICAL"

"The very concise and brief clinical reports that appear in the ARCHIVES each month have been very helpful to me, and *I have found more practical suggestions in them than most of the long and drawn out scientific articles that appear in other journals.*"

"I want to tell you that after subscribing for about 16 years, *I get more practical pediatrics from your journal than from either of the other pediatric journals to which I subscribe.*"

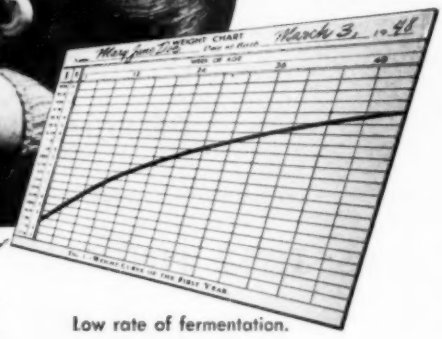
"Because it is *concise and progressive* I consider ARCHIVES OF PEDIATRICS my favorite among pediatric journals. I always look forward with pleasure to the receipt of each number and *glean much of value from it.*"

"The only pediatric journal now on file there (Ellis Hospital, Schenectady) is the American Journal of Diseases of Children and I feel that *yours will be of more practical value to the general practitioners.*"

(*Names on Application*)

ARCHIVES OF PEDIATRICS **45 East 17th St., New York**

To Maintain a Steady Weight Curve



Cartose is a carefully proportioned mixture of dextrins, maltose and dextrose.

Since each of these carbohydrates has a different rate of assimilation, a steady supply of carbohydrate is released for "spaced" absorption.

Low rate of fermentation.

Low incidence of digestive disturbances.

Bottles of 1 U. S. pint.

Write for formula blanks.

Specifically Designed for Infant Feeding

CARTOSE®

MIXED CARBOHYDRATES IN EASY-TO-USE LIQUID FORM

Compatible with all milk formulas

Less Colic · Less Diarrhea

Winthrop-Stearns INC. · NEW YORK 13, N. Y. WINDSOR, ONT.

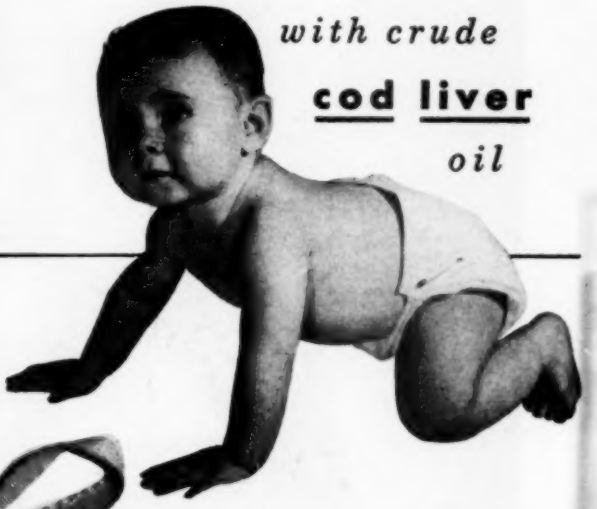
Cartose, trademark reg. U. S. & Canada

the **only** *baby powder saturated*

with crude

cod liver

oil



Desitin®
MEDICINAL and NURSERY
powder

Desitin Powder is a truly different and unique preparation especially formulated for over-all care of the baby's skin. Desitin Powder, being saturated with Crude Cod Liver Oil, does not deprive the skin of its natural fats.

Desitin Powder *soothes, protects, lubricates, heals*

in **diaper rash • intertrigo • exanthema • prickly heat rash**
minor burns • irritations • rashes • chafing

℞ Provides crude cod liver oil (plus its vitamins A and D) in a fine dry dusting base of zinc oxide, talcum, magnesium oxide. In 2 oz. shaker top cans.

requests for professional samples are invited

Desitin CHEMICAL COMPANY • 70 Ship St., Providence, R.I.

• *Makers of Desitin Ointment*

How happy mealtimes influence Nutrition

A BABY'S assimilation of food is influenced by how much he *enjoys* his mealtimes.

When a worried mother asks you how to "make" her baby eat more, help her understand that one of the biggest things she can do for her baby is to avoid mealtime arguments.

Beech-Nut Foods in all their appealing variety are a great help in making mealtimes happy. Their finer flavor arouses *eager* appetite. Your young patient gets a good start nutritionally and emotionally!

A wide variety for you to recommend: Meat and Vegetable Soups, Vegetables, Fruits, Desserts—Cereal Food and *Strained* Oatmeal.

All Beech-Nut standards of production and advertising have been accepted by the Council on Foods and Nutrition of the American Medical Association.

Beech-Nut FOODS for BABIES

Babies love them...thrive on them!

EVERY LISTED PEDIATRIC SPECIALIST

was questioned by an independent research organization about an article published in the Archives of Pediatrics. These specialists were asked whether they agreed with the reprint material.

Of the pediatricians who believed their experience justified an opinion, 156—81.7%—replied yes to all three points in question.

4 out of 5 Leading Pediatricians agree that CREAM OF RICE

a | **gives "more available caloric energy"**
than any wheat, rye, barley, corn or oat cereal. Of the 227 pediatricians answering definitely, 192—84.6%—said yes.

b | **is "more easily digestible"**
than any other kind of cereal. Of the 248 answering definitely, 212—85.5%—said yes.

c | **gives "nutritional energy more rapidly"**
than any other kind of cereal. Of the 220 answering definitely, 178—80.9%—said yes.

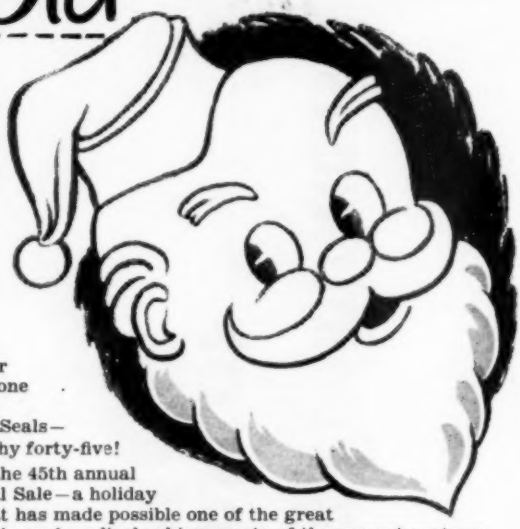
In addition, Cream of Rice is

MOST HYPOALLERGENIC, TOO

As reported in the Archives of Pediatrics by Slobody, Untracht and Hertzmark, "rice . . . shows the fewest allergic reactions of any cereal checked . . . Even children potentially allergic to rice have been shown to tolerate it well when it is cooked in the presence of moisture."

WRITE FOR PROFESSIONAL SAMPLES:
GROCERY STORE PRODUCTS CO., DEPT. AP-10,
WEST CHESTER, PA.

How Old is Santa Claus?



This particular Santa—the one you see on the Christmas Seals—is a very healthy forty-five!

Yes, this is the 45th annual Christmas Seal Sale—a holiday custom that has made possible one of the great social, economic, and medical achievements of the present century.

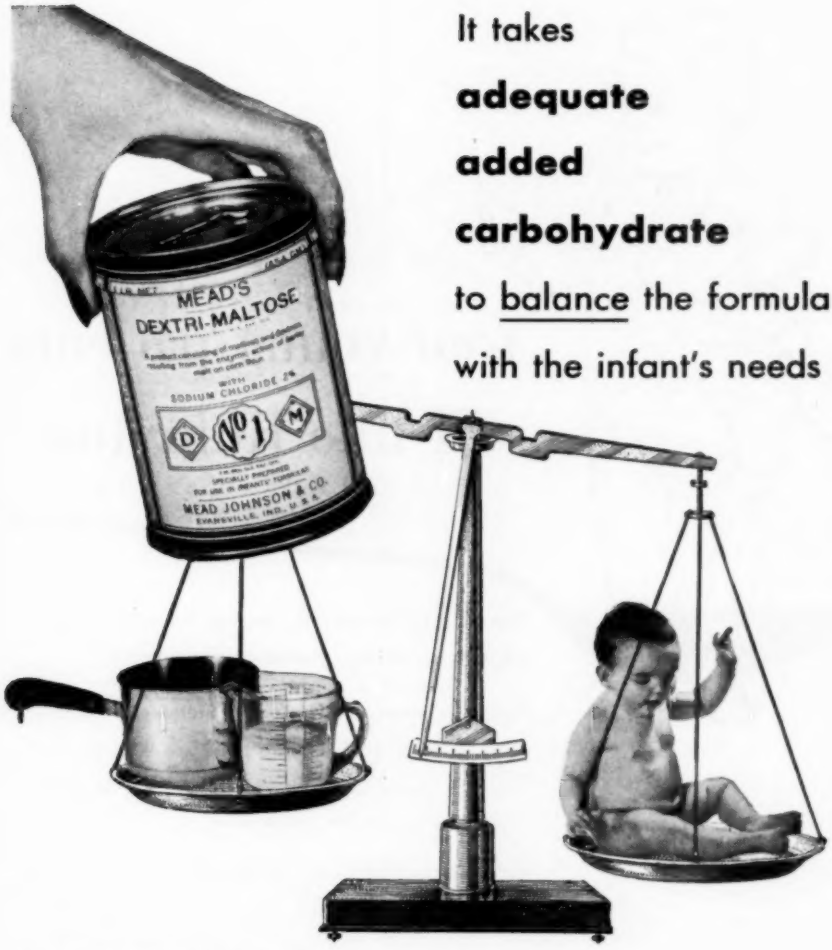
Your purchase of Christmas Seals has helped save 5,000,000 lives. Yet, tuberculosis kills more people than all other infectious diseases combined.

So, please answer once again the call that comes but once a year—and help make possible the campaign against tuberculosis every day of the year.

Because of the importance of the above message, this space has been contributed by

Buy Christmas Seals!

Archives of Pediatrics



It takes **adequate added carbohydrate** to balance the formula with the infant's needs

Added carbohydrate is *required* for well balanced formulas. *Adequate* carbohydrate:

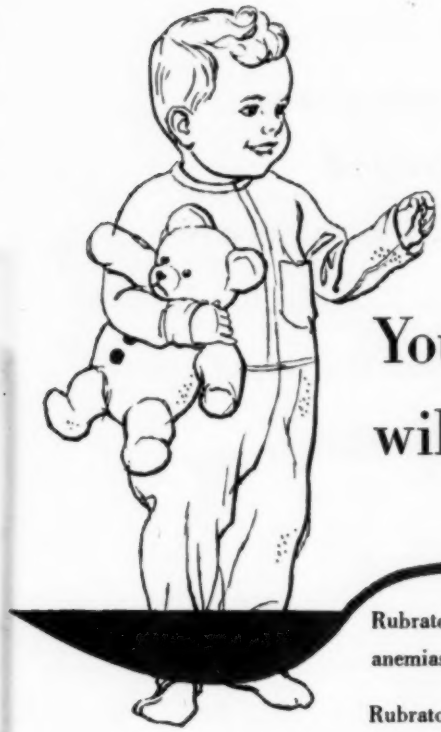
1. Spares protein for tissue building functions.
2. Permits proper metabolism of fat.
3. Promotes optimum weight gain.
4. Encourages normal water balance.

Pediatric authorities recommend a caloric distribution of about 15% from protein, 35% from fat, 50% from carbohydrate. For 40 years, cow's milk and Dextri-Maltose® formulas with this approximate caloric distribution have been used successfully.

These formulas very often consist of ⅓ evaporated milk, ⅔ water and 5% added Dextri-Maltose—about 1 level tablespoon Dextri-Maltose to 5 ounces of formula.

MEAD'S

MEAD JOHNSON & CO.
EVANSVILLE 21, IND., U. S. A.



Your young patients will like Rubraton

Rubraton offers complete therapy for most of the anemias of infancy, childhood and puberty.

Rubraton presents vitamin B_{12}, folic acid and iron in a low alcohol elixir that tastes good . . . straight from the spoon or mixed with water or fruit juice.

'Rubraton' is a Trademark of E. R. Squibb & Sons

Each teaspoonful (5 cc.) of Rubraton contains:

Vitamin B_{12}	4.17 micrograms
Folic Acid	0.28 milligrams
Ferric Ammonium Citrate	220 milligrams

1 or 2 teaspoonfuls t.i.d. Pint bottles

Rubraton

Elixir of B_{12}, Folic Acid and Iron Squibb

SQUIBB MANUFACTURING CHEMISTS TO THE MEDICAL PROFESSION SINCE 1858

ARCHIVES OF PEDIATRICS

VOL. 68 OCTOBER 1951 No. 10

JOHN FITCH LANDON, M.D., Editor

EDITORIAL BOARD

HAROLD R. MIXSELL, M.D., New York
REUEL A. BENSON, M.D., New York
PHILIP M. STIMSON, M.D., New York
JOHN ZAHORSKY, M.D., St. Louis
JOSEPH S. WALL, M.D., Washington
FREDK. H. WILKE, M.D., New York

ACQUIRED HEMOLYTIC ANEMIA OF THE NEWBORN (ERYTHROBLASTOSIS FOETALIS) DUE TO O-B ISO-IMMUNIZATION*

REPORT OF CASE

ABRAHAM M. FRUMIN, M.D.

AND

ALBERT KOHN, M.S.

Philadelphia.

The role of iso-immunization to the A and B agglutinogens in producing fetal and neonatal morbidity and mortality had not been recognized earlier since the manifestations of the disease were usually mild. In typical cases the baby developed mild jaundice and a moderate anemia, usually on the first or second day of life. The disease was then arrested and recovery usually ensued without treatment in four to five days, although in more severe cases blood transfusions might be required.

This syndrome has been designated "icterus precox" by Halbrecht[1] in order to distinguish it from the so-called physiologic icterus or icterus neonatorum. Since then case reports have been written by Polayes[2-5], Wiener[6-8], Grumbach[11], Tortora[12], Levine[13, 14], Weisl[15] and Robinson[17], bringing attention to this disease.

Most of the cases have remained undetected, except for the presence of a moderate anemia. This has been, for the most part, disregarded as to its possible relationships to the A-B-O immunologic

*From the Department of Research, Mt. Sinai Hospital, Philadelphia.
Assistant in Pathology (Hematology), Mt. Sinai Hospital, Instructor in Medicine, Woman's Medical College of Pennsylvania (Dr. Frumin).
Serologist, Mt. Sinai Hospital, Philadelphia (Mr. Kohn).

systems. The clinical role of Rh factors in causing the acquired hemolytic anemia of the newborn have been stressed sufficiently to make one alert to its significance. Besides, the often dramatic onset and development of symptoms and signs were sufficient to draw attention to the disease. For the most part, however, A-B-O incompatibilities have been only mild or moderate in severity and the clinical course less pronounced.

The authors wish to report a case illustrating O-B incompatibility with a moderately severe anemia which was rather markedly protracted in its duration.

CASE REPORT

This white female child was delivered by low forceps of a gravid II para O mother on November 20, 1949 at 1:52 A.M. on the Obstetrical Service of the Mount Sinai Hospital. The child measured 19 inches in length and its head circumference was 12⅝ inches at birth. Its initial birth weight was 6 pounds 3 ounces.

Physical Examination was essentially normal. The color was good and the cry was normal. No evidence of any hepatosplenomegaly was noted. The initial blood count on November 21, 1949 at 9 A.M. revealed the following values: Red blood cells 4,530,000, hemoglobin 18.1 grams (116 per cent), white blood cells 22,100 with 3 per cent bands, 63 per cent segmented polymorphonuclear leukocytes, 32 per cent lymphocytes, 1 per cent monocytes, 1 per cent eosinophiles. Examination of the blood smear showed a "double-population" of erythrocytes. This was characterized by the presence of many polychromatophilic macrocytes and spherocytes. Five nucleated red blood cells per 100 white blood cells were counted.

Later in the same day the child was noticed to be clinically jaundiced with a definite scleral icterus being present. No evidence of an enlarged liver or spleen was demonstrable. At 10 P.M. the hemoglobin had fallen to 15.9 grams.

On November 22, 1949 at 9 A.M. another blood count showed the following values: Red blood cells 3,320,000, hemoglobin 15.1 grams, white blood cells 16,700 with 1 per cent metamyelocytes, 8 per cent bands, 47 per cent segmented polymorphonuclear leukocytes, 34 per cent lymphocytes, 3 per cent eosinophiles, 1 per cent basophiles, 6 per cent monocytes. Examination of peripheral

smear showed many polychromatophilic macrocytes and spherocytes. One nucleated erythrocyte per 100 white cells was counted.

At 4 P.M. of the same day a moderate degree of pallor was noted for the first time. The red cell count had fallen to 2,900,000 and the hemoglobin had dropped to 13.1 grams. A reticulocytosis of 10.4 per cent was obtained.

Because of this sudden and precipitous fall in the red cell count and hemoglobin, the child was given 65 cc. of whole blood. The child responded well to this transfusion. On November 25, 1949 the following values were obtained: Red blood cells 4,480,000, hemoglobin 16.8 grams (108 per cent), white blood cells 10,100 with 46 per cent segmented polymorphonuclear leukocytes, 54 per cent lymphocytes.

The child was discharged from the hospital but was examined periodically by her attending pediatrician. A gradual developing pallor and increasing anemia developed so that further hospitalization was required on December 16, 1949. The following values were found: Red blood cells 2,640,000, hemoglobin 10.9 grams (70 per cent), reticulocytes 1.4 per cent, platelets 210,000 per cumm., white blood cells 16,200 with 6 per cent bands, 20 per cent segmented polymorphonuclear leukocytes, 66 per cent lymphocytes, 7 per cent eosinophils, 1 per cent monocytes. The smear showed a moderate amount of polychromatophilic macrocytes and some few spherocytes.

Unfortunately the child was removed from the hospital by its parents against the advice of the physician. A recent conversation with the pediatrician elicited the information that normal values for the red cell and hemoglobin had been attained by April 1, 1950.

Immunologic Studies. Careful immunologic studies were done in an attempt to discover the etiology of the hemolytic process. The studies on the family revealed the following:

(1) *Blood Grouping*

	ABO	D	C	E	c
Father	B	+	+	+	+
Mother	O	+	O	+	+
Baby	B	+	O	+	+

(2) *Circulating Antibodies.* Attempts to demonstrate a cir-

culating antibody present in either saline or albumin solution at temperatures of 37°C, 21°C, or 3°C were fruitless. No agglutinin or hemolysin was demonstrable. This ruled out any incompatibility as far as the CDE system and negated the possibility of an abnormal circulatory antibody in the mother's serum.

(3) *Coombs Test.* The Coombs test to determine the presence of a fixed antibody on the patient's red cells was done. A weekly positive result was obtained. The Coombs test, in our hands, has been of limited importance in determining the presence of an acquired hemolytic anemia. When a positive result is obtained it is of corroborative importance but we have seen instances of an acquired hemolytic anemia in which the Coombs test was negative.

(4) *Anti-B-titre of the Mother's Serum.* Titration of the mother's serum was performed against group B red cells and agglutination was found to be present in a titre of 1:640. Titration of the mother's serum against A cells revealed a value of 1:320. This indicated a specific increase for the B factor. Apparently the hemolytic process in the child was due to iso-immunization of the mother's serum by the red cells of the fetus.*

DISCUSSION

Acquired hemolytic anemia of the newborn, or erythroblastosis foetalis, was given its important place in pediatric hematology by the contribution of Levine and Wiener in their studies on the Rh factor. This type of anemia was often dramatic in onset, poor in prognosis, and the mortality in untreated cases unusually high.

Acquired hemolytic anemia of the newborn due to A-B-O incompatibility has only recently become more important to the obstetrician and pediatrician. These cases have been milder in intensity though their duration was much longer than previously suspected.

Clinically these patients showed pallor as a more marked finding than jaundice. Enlargement of the liver and spleen was extremely rare. Edema of the skin and subcutaneous tissues, or hydrops foetalis, were never found. Because of the mild to moderately severe degree of anemia little attention had been previously focussed on the A-B-O system of blood grouping.

*A specimen of blood was sent to Dr. Philip Levine of the Ortho-Research Foundation who confirmed our findings and corroborated our diagnosis.

The blood picture in these cases showed the usual findings of a hemolytic anemia. There was an anemia which was normocytic and normochromic in nature. Reticulocytosis was apt to be present. In our case it reached a value of 10.4 per cent. A leukocytosis accompanies this anemia and there may be either a polymorphonuclear leukocytic or lymphocytic response.

A careful study of a well prepared peripheral smear would show the usual findings in an acquired hemolytic anemia. Two types of erythrocytes were found in abnormally high numbers. The first of these, a polychromatophilic macrocyte, is the earliest nonnucleated erythrocyte, beyond the reticulocyte stage. The other is the "oldest" type of red cell, the spherocyte. The occurrence of these two types of erythrocytes in the peripheral smear should lead one to strongly suspect a hemolytic process.

Immunologic studies were of diagnostic importance in the determination of the type of immunologic system that was involved. The importance of blood grouping and subgrouping is obvious. The determination of the nature of the circulating antibody entailed careful studies. These involved not only the suspension of erythrocytes in saline and albumin but likewise serial determinations carried out at varying temperatures, i.e., 37°C, 22°C, and 3°C. By these procedures the presence of an abnormal circulating antibody can be determined.

The Coombs test[16] has been described as a valuable aid in the differential diagnosis of a congenital and an acquired hemolytic anemia. In our hands it has been a useful adjunct to the diagnosis of acquired hemolytic anemia.

The presence of a positive history of previous immunization is naturally of vital importance. This may occur either through previous pregnancies or through prior blood transfusions. Pregnancies need not necessarily have been full term and blood need only have been given intramuscularly to produce the initial sensitization. In our patient the prior pregnancy had terminated in a miscarriage which evidently was the necessary sensitization required to produce the hemolytic anemia of the newborn at this time. The importance of a careful immunologic survey in a newborn child with a hemolytic anemia cannot be too strongly stressed.

The type of anemia present precludes any advantageous result from the use of either liver extract, folic acid, ferrous sulfate or

B_{12}. Since we were dealing with what was essentially a hemolytic process, whole blood transfusions were the only effective therapeutic agents. As in our case, the clinical course may not be dramatic and the prognosis not poor. Nevertheless, in these patients, a protracted time interval developed before the correction of the anemia was successful.

SUMMARY

(1) A case of hemolytic anemia of the newborn due to O-B incompatibility is presented.

(2) An enumeration of the various methods, hematologic and immunologic, necessary for the evaluation of the patient is offered.

REFERENCES

1. Halbrecht, I.: Role of Hemagglutinins Anti-A and Anti-B in Pathogenesis of Jaundice of the Newborn—Icterus Neonatorum Precox. Am. J. Dis. Child., 69: 208-209, 1944.
2. Polayes, S. H.: Erythroblastosis Foetalis Unrelated to the Rh Factor; Iso-Immunization of Group O Mothers by Group A Children. Meeting N. Y. Path. Soc., Jan. 1942; Nov. 29, 1945.
3. Polayes, S. H.: Erythroblastosis in Mothers with Rh Positive Blood. Am. J. Dis. Child., 69: 208-209, 1944.
4. Polayes, S. H.: Erythroblastosis Foetalis Unrelated to the Rh Factor. Am. J. Clin. Path., 15: 467-470, 1945.
5. Polayes, S. H. and McNally, J., Jr.: Iso-immunization with the A and P Factors and Its Relation to Hemolytic Disease of the Newborn. Am. J. Clin. Path., 18: 375-386, 1948.
6. Wiener, A. S.: Recent Advances in Knowledge of the Rh Blood Factors with Special Reference to the Clinical Application. Tr. & Studies Coll. Phys. Phila., 105-122, 1945.
7. Wiener, A. S.; Sonn, E. B and Hurst, J. G.: Pathogenesis of Erythroblastosis Foetalis III, Illustrating Case Histories of A-B Sensitization. Studies on Individual Differences in Human Blood and Their Practical Applications. Paper No. 1, Brooklyn, N. Y., July 15, 1946.
8. Wiener, A. S. and Sonn, E. B.: Permeability of the Human Placenta to Iso-antibodies. J. Lab. & Clin. Med., 31: 1022-1024, 1946.
9. Jonsson, B.: Acta path. et microbiol. Scandinav., 13: 424, 1936.
10. Diamond, L. K.: Quoted in Weisl.
11. Grumbach, A. and Gasser, C.: A-B-O Inkompatibilitaten und Horbus Haemolyticus Neonatorum. Helvet. paediat. acta, 3: 447-474, 1948.
12. Tortora, M.: "Icterus praecox" da Sensibilizzazione Materna versa Il Fattore A. Arch. obstet. e. ginec., 52: 436-438, 1948.
13. Levine, P.: Genetic and Constitutional Causes of Fetal and Neonatal Morbidity. Ann. New York Acad. Sc. 46: 939-961, 1946.
14. Levine, P.: Serological Factors as Possible Causes of Spontaneous Abortion. The Abortion Problem. Chap. VI, 1944.
15. Weisl, B. A. G.: Possible Hemolytic Anemia of the Newborn Due to High Titre Anti-"A" Agglutinins in a Group "O" Mother. Am. J. Obst. & Gynec., 56: 1002-1004, 1948.
16. Coombs, R. R. A.; Mourant, A. E. and Race, R. R.: A New Test for the Detection of Weak and "Incomplete" Rh Agglutinins. Brit. J. Exper. Path., 26: 255, 1945.
17. Robinson, G. C.; Phillips, R. M. and Prystowsky, M.: Spherocytosis and Increased Fragility Occurring in Erythroblastosis Foetalis Associated with A-B-O Incompatibility.

CONGENITAL MEGACOLON*

Report of Case

Thomas I. Longworth, M.D.
New York.

Herein is reported a moderately severe case of congenital megacolon. All degrees of severity are possible but more commonly the disease produces a chronic progressive dilatation of the affected colon with constitutional signs and symptoms. This particular child has an eleven and one-half year history of constipation and obstipation with no or only temporary results from a long medical regime. Surgery was finally performed; at first, a sympathectomy with poor results and later a resection based upon a new etiological concept. The final surgical result of this case, following the treatment of Swenson, shows much promise as a permanent cure to a previously poorly treated disease.

CASE REPORT

A three-year-old colored male of French-Negro parentage was first admitted to Metropolitan Hospital on May 22, 1942. The chief complaint was postprandial vomiting, generalized abdominal pain, and a palpable mass in the right lower quadrant, all of two days duration. The mass was previously noted by the mother one year before admission but subsided spontaneously. The history revealed a chronic constipation since the child was six months old. Various laxatives and suppositories were used almost daily in order to evacuate the bowels. The patient, a second child, was a full term baby delivered spontaneously with no complications. Birth weight of nine pounds. He had an apparently normal development; first tooth at six months, walked at eleven months. Childhood illnesses included measles and chickenpox at two years, and whooping cough at three years. No operations or accidents. His appetite had always been good, slept well, but not as active as other children his age.

Physical examination revealed a fairly well developed, slightly undernourished three-year-old colored male in no acute distress. Examination was normal except for large injected tonsils, a palpable, doughy, moveable mass in the right lower quadrant which filled the right flank, and hard impacted feces filling the rectum.

*Submitted as partial fulfillment of the course in Senior Pediatrics at the New York Medical College, Flower and Fifth Avenue Hospitals.

The impression at this time was (1) impacted feces from chronic obstruction (2) hypertrophic tonsillitis. Many attempts of digitally breaking up the fecal mass plus several oil retention enemas were necessary before adequate evacuation with reduction of the mass was attained. A trial of syntropan of gradually increasing dosage from 5 mg. t.i.d. to 50 mg. t.i.d. (covering a two months period) was not helpful. The child was then placed on acetyl choline, 0.03 gm. t.i.d. and gradually increased to 2 gm. per day with no results. Mecholyl and vitamin B_1 were also tried but had to be discontinued due to toxic symptoms. A barium enema x-ray report revealed a marked redundancy of the sigmoid with dilation of proximal half. A proctoscopic examination revealed no pathology. A joint pediatric-surgical conference decided that the most probable diagnosis was congenital megacolon; surgery deferred. During the six month hospitalization the child had occasional bowel movements but the amounts were very small. Laxatives and enemas every two days were necessary for comfort. The patient was discharged with instructions to mother to use mineral oil daily and enemas as seemed necessary and to be followed in the clinic.

The child attended follow-up clinic four times following discharge but did not return after April 13, 1944.

The Second Admission occurred on July 14, 1941 at the age of six years. For six days previous to admission the child had no bowel movements. Daily enemas, castor oil, milk of magnesia produced only one fragmentary stool and the patient vomited three times on the day of admission. Since first admission the child required daily mineral oil and enemas every other day.

Physical examination revealed a thin, lethargic, poorly developed and poorly nourished colored male boy of six years. A slight pigeon chest; a distended (mainly right sided) abdomen, non-tender; hard feces within the rectum were found. The temperature, pulse and respiration were within normal limits. The laboratory reported: hemoglobin 12 gm.; R.B.C. 5.5 million; W.B.C. 14,000, 80% polys, 15% lymphs, 5% monos; blood urea nitrogen 11.0 mg.%; creatinine 1.0 mg.%; nonprotein nitrogen 22 mg.%; glucose 82 mg.%. Gradual reduction of abdominal distention and emptying of the large bowel were obtained with oil retention enemas, soapsuds enema and sodium bicarbonate enemas. A trial of syntropan, 50 mg. b.i.d. and prostigmine, 0.5 cc. per day, were ineffectual. Vitamin B_1

35 mg. per day with mineral oil seemed satisfactory. The child was discharged after one month with instruction to use vitamin B_1 and mineral oil. Discharge diagnosis was congenital megacolon.

The Third Admission occurred on March 22, 1946 for acute follicular tonsillitis. Since previous admission the child had been taking one enema per day; appetite had been poor. Physical examination revealed a pale, undernourished, lethargic colored boy. A baseball sized mass was present in the right lower quadrant; no feces within the rectum. Enemas produced a gradually increasing return. Trials of syntropan, prostigmine, and mecholyl failed. Barium enemas revealed an enormously enlarged sigmoid, widened, elongated, and atonic. Radiological diagnosis was Hirschsprung's disease. The child was signed out by the mother.

The child was admitted for the *fourth time* on October 1, 1948 at the age of 9½ years. He had had no return from enemas for six days and three episodes of vomiting following meals with abdominal cramps one day before admission. He had previously been maintained on enemas and mineral oil. On physical examination appeared thin and chronically ill. The abdomen was distended, loops of small bowel were visible and palpable through the abdominal wall, tympanitic to percussion, prominent superficial abdominal veins, no definite masses, slight generalized tenderness with rebound tenderness, few rumbling peristaltic sounds, impacted feces in rectum. Impression was intestinal obstruction due to fecal impaction with megacolon. The patient was treated with Miller Abbott tube with continuous suction, digital removal of fecal impaction, then oil retention enema followed with soapsuds enema, parenteral fluids, prophylactic penicillin. The child responded well to treatment but developed a right lower lobe pneumonia which was treated with penicillin. A spontaneous bowel movement occurred six days after admission. Protoscopic and sigmoidoscopic examination revealed no pathology except for dilation of the sigmoid. A course of 25 mg. of mecholyl b.i.d. resulted in two normal bowel movements per day without the aid of laxatives or enemas. Barium enema revealed no essential change since the 1946 admission. The child was discharged after a two-month stay to the clinic with instructions to take mecholyl, 50 mg. b.i.d.

The Fifth Admission occurred on March 10, 1949 because of a four-day history of ineffectual enemas. The child had been maintained on enemas and mecholyl.

Physical examination revealed a fairly well nourished 90 pound colored boy with a baseball sized mass in the right abdomen and fecal impaction. Oil retention and H_2O_2 enemas were only partially effective. Anesthesia was necessary to digitally break up the fecal mass. A joint conference decided it was time for surgical intervention. A barium enema under spinal anesthesia revealed good contractions of the sigmoid. On this basis a two stage bilateral lumbar sympathectomy was performed without difficulty. A postoperative barium enema x-ray showed the previously described redundancy and hypertrophy of the sigmoid with no appreciable alteration in the lumen. Immediately before discharge, 3½ months after admission, a barium enema revealed a slight diminution in the calibre of the colon. The child was discharged to the clinic on mineral oil.

On February 24, 1951 the child was admitted for *the sixth time*, now at the age of twelve years. The complaint was malaise, anorexia, and progressive abdominal distention. Since 1949 he had been maintained on various laxatives and twice weekly enemas. The patient was fairly well developed and nourished.

Physical examination was negative except for: a distended, nontympanitic abdomen, doughy mass in entire lower abdomen and left flank with moderate tenderness, hard impacted feces in rectal ampulla. Enemas and colonic irrigations were effective and the mass reduced; sigmoidoscope showed no areas of constriction. Laboratory findings: hemoglobin, 11.5 gm.; R.B.C., 4.07 million; W.B.C. 12,200, 67% polys, 30% lymphs, 2% monos, 1% eosinophils; total serum protein 6.5, albumin 4.1, globulin 2.4; albumin-globulin ratio 1.7. A surgical conference decided that more radical surgery was indicated. This would follow the technique as recommended by Swenson, which means resection of the involved portion of the rectum and the dilated portion of the sigmoid with a primary anastomosis by pull through technique. The patient was prepared with sulfasuxidine, penicillin, streptomycin, laxatives and colonic irrigations. At operation the rectosigmoid from 4 cm. above the anus to just above the junction of the colon and sigmoid was resected. An end-to-end anastomosis was done with carefully placed interrupted sutures by a pull through technique. The postoperative course was complicated by one episode of urinary retention, a spiking fever of 103° F. with left lower quadrant pain.

Treatment with chloromycetin, Miller Abbott intubation and

intravenous fluids were started. A dramatic drop in temperature and relief of pain occurred in 24 hours. It was believed that the patient had a volvulus with spontaneous reduction. An operative barium enema showed no abnormalities of the large bowel. The pathological report of the surgical specimen was as follows: 18 cm. long large intestine, 5 cm. in diameter, walls thickened with a hyperplastic mucosa with irregular rugae and pseudopolypoid formations. Early in the postoperative course the child had 4 to 10 liquid to soft bowel movements per day without sphincter control. He gradually developed control and on discharge, April 11, 1951, he had an average of four soft, poorly formed bowel movements per day with good control. He is now being carefully followed in the clinic. The child now appears more alert, appetite is good, 3 to 4 soft formed bowel movements per day requiring no laxatives or enemas, sphincter control is good and there is no evidence of incomplete bowel movements.

DISCUSSION

The etiology of megacolon may be placed in two general catagories: 1. Acquired or partially obstructive lesion. 2. Congenital or idiopathic. Since all forms of mechanical obstruction have been ruled out, we are dealing with, in this particular case, a form of congenital megacolon, or more specifically, megasigmoid. Many etiological factors have been presented as the cause of congenital megacolon but as yet no uniform agreement has been reached. Imbalance of the autonomic nerve supply to the affected part has been a popular etiology in the past. This may be due to an excessive or diminished parasympathetic activity or an excessive or diminished sympathetic activity. This supposition was the result of the fair to good therapeutic recovery with the use of sympathectomies, sympathetic and parasympathetic stimulation, and paralyzing drugs. Some authors favor the congenital absence of enteric nerve cells in the dilated portion. Swenson, more recently and with much support, has demonstrated the fact that a distal constricted portion proximal to the anus is the primary site of pathology. The dilated portion proximal to the area is a secondary result and is normal except for compensatory dilation and hypertrophy. Histological examination of the constricted portion, readily demonstrated on x-ray by special technique, shows an absence

of nerve ganglia of the intramural plexus. The histology of the dilated portion reveals a normal nerve supply. Swenson's operation consists of resection of the rectum and rectosigmoid with preservation of the sphincters. He has attained excellent results with a probable permanent cure in a relatively large series of cases. Bowel control gradually becomes normal in every respect. Swenson's treatment for congenital megacolon shows greater promise for permanent cure of this chronic relapsing disease than any other regime, and should be considered in all cases of severe megacolon.

BIBLIOGRAPHY

1. Mitchell and Nelson: Textbook of Pediatrics, 4th Ed., p. 638.
2. Brennemann: Practice of Pediatrics, Vol. III, Chap. 7, p. 10.
3. Robbin, Lewis: Congenital Megacolon in Young Infants and Children. ARCH. PEDIAT., 64: 459-475, Sept. 1947.
4. Swenson, O., and Bill, A.: Resection of Rectum and Rectosigmoid with Preservation of the Sphincter for Benign Spastic Lesions Producing Megacolon. Surgery, 24: 212, Aug. 1948.
5. Swenson, O.; Rheinlander, H., and Diamond, I.: Hirschsprung's Disease: A New Concept of the Etiology. New England J. Med., 241: 551, Oct. 13, 1949.
6. Hersh, J.: Congenital Megacolon—Hirschsprung's Disease. Am. J. Surg., 74: 815, Dec. 1947.

TREATMENT OF YAWS BY AUREOMYCIN. (Trans. Royal Soc. Trop. Med. and Hyg., London, 44: 315, Dec. 1950). Five children, 3 to 8 years of age, with florid secondary yaws were treated with aureomycin hydrochloride. This was given in the form of capsules by mouth, 250 mg., three times daily for seven days. This dose cleared up the secondary lesions and prevented recurrence for at least six months. Aureomycin given by mouth proved also of value in two children, 8 and 10 years of age, respectively, with late secondary or early tertiary yaws. They were given 500 mg. three times daily for seven days. It appears that in late secondary and tertiary yaws aureomycin in the dosage given is less spectacular in its action than in patients with a florid secondary skin eruption. Nevertheless, it is of significant value in healing chronic ulcers and in relieving the pain in yaws periostitis. The oral administration of aureomycin caused no toxic symptoms such as fever, headache, nausea, vomiting or skin eruptions. Aureomycin behaves in yaws much in the same way as does penicillin. It has the advantage over penicillin in that it is effective by mouth in comparatively small doses. In mass treatment this is obviously a great advantage, since besides the pain of daily intramuscular injections there is a great saving of labor, apparatus and time.—*Journal A.M.A.*

NEUROBLASTOMA*

Report of Case

Myron B. Harkavy, M.D.
New York.

With the development of more refined techniques, tumors of the sympathetic nervous system are being diagnosed earlier, with greater frequency, and with greater accuracy. These tumors vary in their gross and histological characteristics depending upon the stage of development at which neoplastic growth begins. The primordial cells, which are derived from neural crest material, are multipotent and may develop in one of several directions. Thus, via sympathoblasts (neuroblasts) they may develop into sympathetic nervous cells, via spongioblasts or lemmoblasts into peripheral glia, and, finally, via pheocromoblasts into the pheochrome (chromaffine) cells of the adrenal, the carotid body and other organs. Tumor formation may occur at any stage (Bailey and Cushing) and, therefore, in keeping with this hypothesis, Scott, Oliver and Oliver (1933) suggested the following classification[1]: (1) Entirely undifferentiated tumors which include sympathiocoblastomas. (2) Incompletely differentiated tumors which include sympathetic neuroblastomas, spongioblastomas and pheochromoblastomas. (3) Completely differentiated tumors which include ganglioneuromas, and pheochromocytomas.

Emigration of neural crest material throughout the body to form various parts of the sympathetic system continues till after puberty (Wiesel, 1905) and the "long distance which these cells have to travel favours faulty cell development so that immature groups of cells may be left behind which are potentially capable of proliferation."[1]

Neuroblastomas, also labeled sympathicoblastomas and sympathoblastomas, fall into the first category listed above and are probably the most common abdominal malignancies of childhood, being one of the four most frequent malignancies in children. Mandeville[2] writes of 431 known cases up to 1947 and feels many more were misdiagnosed as other lesions. The tumor was first described by Virchow (1864) under the name of glioma, but it was not until

*Submitted as partial fulfillment of the requirements of the course in Senior Pediatrics at the New York Medical College, Flower and Fifth Avenue Hospitals. The author is at present interning at Beth Israel Hospital, New York City.

1910 that Wright established its origin from anlage of the sympathetic nervous system. He first used the term neuroblastoma to describe them. The tumor may arise in any part of the body, having been reported in the ciliary ganglion, cervical sympathetic trunk, thorax, celiac ganglion, small intestine, uterus, sacral plexus, skin, along peripheral nerves,[1] scapula, mediastinum and intervertebral foramen.[2]

Presenting signs and symptoms may include ecchymosis of one or both lids, bluish discoloration of the temporal regions with a definite palpable mass, exophthalmus, papilledema, and roentgenological evidence of bone destruction in the skull with separation of the sutures and involvement of the long bones. Other findings are palpable abdominal masses with rapid increases in size, anemia, cachexia and weakness.[3] Hutchinson and Pepper have described different clinical pictures based on different routes of metastases as seen in tumors of the right as opposed to that of the left adrenal. This concept is not widely accepted at the present time.

The following case history is presented to show how the presenting symptom may be entirely different from the usual course as described above, how widespread metastases may be present before serious symptomatology occurs, and how dramatic, although extremely transient, improvement may occur under present day therapy.

CASE REPORT

J. W., a 4-year-old white male, was admitted to the hospital pediatric ward because of constipation of two weeks duration and vomiting for three days prior to admission. He had been in good health until two weeks before admission at which time severe constipation developed, failing to respond to the usual laxatives and enemata. Malaise, listlessness, lethargy and pallor gradually became evident. Three days before admission severe vomiting occurred, with intensification of the above mentioned symptoms.

Positive Physical Signs on Admission included a stiff neck, a positive Kernig sign, and multiple small palpable masses in the abdomen. Other neurological signs were negative. The impression at this time was that the child had either a meningitis or encephalitis. However, x-rays were taken and revealed many osteolytic lesions scattered throughout the pelvis, skull, vertebrae and humeri.

This moth-eaten appearance was thought suggestive of neuroblastoma, the diagnosis being further aided by the appearance of calcification in the right adrenal area. Laboratory findings included hemoglobin 12 grams, W.B.C. 10,000, blood urea nitrogen 25 mgms., blood chlorides 918 mgms., albumin 4.4 grams, globulin 2.5 grams, potassium 3.4 mEq., sodium 131 mEq. Bone-marrow aspiration showed the typical rosette formation of neuroblastoma.

Within several days right external rectus palsey developed as did a severe frontal headache. The right knee jerk disappeared and a positive Babinski was elicited on the same side. The patient then lapsed into coma. These latter symptoms were thought to be due to a cerebrovascular accident with hemorrhage into the left cortex.

At this point the patient was transferred to Memorial Hospital where, still in coma, he was given 6 mgms. of nitrogen mustard intravenously. Hypertension developed on the following day (158/128) and one day later there appeared ecchymosis of the right orbit with ptosis and proptosis. On continued nitrogen mustard therapy the blood pressure dropped, the headache disappeared, and the child became sufficiently alert and rational to be discharged to the clinic 20 days after admission.

Nine days later, the child was readmitted with a recurrence of his original complaints. Repeated therapy with nitrogen mustard gave only fair results. A progressive downhill course developed, and at this writing the child was failing rapidly.

Treatment, as noted, may offer only slight palliation. Tumors of this type are moderately radioresponsive to the earliest radiation but resistance increases with subsequent treatment.[2] The nitrogen mustards do not always produce the dramatic results as in the case cited, but they, as well as the antifolic acid compounds, are receiving intensive study in the therapy of this as well as many other neoplastic diseases.

Prognosis is very poor, a fatal outcome being expected by some in an average of four months,[3] by others in from two to five years.[1] Occasional instances are reported in which proven cases of neuroblastoma have not been fatal. One reported case is in excellent health five years after operation.[4] Cushing and Wholbach (1927) and Févre, Lamy, and Baudoin (1943) reported similar cases. Rarely, the malignant lesion may differentiate to such a degree as to become a benign ganglioneuroma.[1]

Diagnosis is based upon the clinical picture plus histologic identification of the typical cells which are sympathoblasts. These usually are associated with delicate fibrils which may be arranged in parallel bundles. One or more rows of these cells may be formed about a central mass of fine fibrils forming the characteristic rosette.[5] On x-ray, lytic lesions may be seen as well as productive changes with radiating spicules as described by Rypins. Calcification in the tumors, if noted in the roentgenogram, may be helpful as an aid in preoperative diagnosis. Calcification may vary from a fine uniform stippling to irregular confluent shadows.[6] Histologic confirmation is, of course, necessary for definite diagnosis. This is not too easy in neuroblastoma as the cells frequently are confused with those of lymphosarcoma and small cell carcinomas. In one series of cases, after a review of the tissue sections of so-called neuroblastomas, one was found to be a Ewing's sarcoma, one a dysgerminoma, and one a primary bronchogenic carcinoma.[2] Murray and Stout[7] report a new diagnostic method utilizing in vitro cultures of tumor tissue in which neurofibril development and other distinctive characteristics of the cells may enable the diagnosis to be made in as little as 24 hours.

SUMMARY

A case report of neuroblastoma in a 4-year-old male child has been presented with a brief review of the embryologic pathogenesis of the tumor, its usual clinical symptomatology, its prognosis, treatment and diagnosis.

REFERENCES

1. Olesin, H., and Sjontoft, F.: Sympathicoblastomas with Metastases to the Orbit. Acta ophth., 26: 67, 1948.
2. Mandeville, F. B.: Sympathoblastoma (Neuroblastoma), Adrenal, Abdominal and Mediastinal. Urol. & Cutan. Rev., 53: 403, 1949.
3. Loomis, E. A., and Loomis, G. L.: Adrenal Sympathoblastoma (Neuroblastoma). Minnesota Med., 32: 606, 1949.
4. van Creveld, S., and van Dam, R.: Prognosis in Sympathoblastoma. Acta paediat., 343: 279, 1947.
5. Chandler, F. A., and Norcross, J. R.: Sympathicoblastoma. J.A.M.A., 114: 112, 1940.
6. Mandeville, F. B.: Calcification in Sympathoblastoma (Neuroblastoma). Radiology, 53: 403, 1949.
7. Murray, M. R., and Stout, A. P.: Distinctive Characteristics of the Sympathicoblastoma Cultivated in Vitro. A Method for Prompt Diagnosis. Am. J. Path., 23: 429, 1947.

A PEDIATRICIAN VIEWS THE TRENDS IN CHILD PSYCHIATRY*

JEANNE SMITH, M.D.
New York.

Some of the developments of modern psychiatry which require critical evaluation at this time are the new methods of psychiatric procedures practiced on children. They are insulin and metrazol shock, electric shock and the latest method of somatic psychiatric treatment, prefrontal lobotomy. Children as young as three and four years old are being treated with these methods. What is their scientific rationále? What are the diagnostic criteria on which they are based, and what attitude should the pediatrician take with regard to them?

As in any other clinical study in pediatrics, one must examine the indications for the procedures, the symptomatology of diseases in which it is recommended and used, and, most important, the prognosis with and without these procedures. For whether it is a question of a child's physical or mental life, the least one must know is a rational basis for prognosis.

The largest series of cases of children receiving electric shock therapy shall be reviewed here, with a view to determine the indications and to analyze the results of such treatment.

Bender[3] reports on 100 cases which were diagnosed as "Childhood Schizophrenia." Childhood schizophrenia, according to Bender's definition, is ". . . a clinical entity, occurring in childhood before the age of eleven years, which reveals pathology in behavior at every level and in every area of integration or patterning within the functioning of the central nervous system, be it vegetative, motor, perceptual, intellectual, emotional or social."

A further elaboration of this disease entity is considered. "As to the age of onset, there are three critical periods. The first two years of life not uncommonly includes the onset of a mental illness in children which subsequently proves to be schizophrenia. The development is reported by the mother to have been uneven and there may be no point at which the child appeared normal and then regressed. . . . From three to four and a half years is the most common period of onset. This group is more readily recognized because of the normal devlopment up to this period. The regression may be

* From the Department of Pediatrics, New York Medical College, Flower and Fifth Avenue Hospitals and Metropolitan Hospital, New York.

insidious and very slow, or abrupt and very rapid, so that in 6 months the child may lose all he has gained in 3 years, especially the socially oriented behavior patterns, leaving him well developed physically with good gait and exaggerated graceful motility, but having lost many of his acquired habit patterns, language, object relationship, with increased anxiety and physical dependence on the mother. The child may show increased activity with anxiety, emotional instability, irritability, excitement, unprovoked aggression (usually oral) and temper tantrums with fears, phobias, disturbed sleep, accelerated language development, and considerable preoccupation with all of the psychological problems of identity, body functions, physical and social orientation. There will be an aggressive dependence on the mother and all adults. In general, those children with onset of schizophrenia before age five, who show severe interference or regression in fundamental habit patterns, language, object relationship, and motility, and show a loss of anxiety, are the most severe cases. In our experience they do not indicate a tendency to remissions nor do they respond much to shock treatment. The third common childhood period of onset is the prepuberal period age 10 to 11½. In these children the clinical picture is quite different. With the obsessional-compulsive thinking added to the psychological problems of the schizophrenic child with his disturbance in identification and orientation, he evolves persecutory systems in which he suspects that either he or his parents are changed and that other children are against him because he cannot identify with them. Inability to keep track of the periphery of his body leads to a breakdown in personal habits and care. In some cases the behavior is like delinquency; the child truants, wanders away from home and enters strange dwellings in an effort to orient himself."

There are other descriptions in this article of the author's criteria for the diagnosis of schizophrenia. "The presence of a severe anxiety in a child, unaccounted for by a reality situation, is in itself suggestive of schizophrenia. When schizophrenia occurs in the first few years or before language is well established, there is usually more or less retardation, inhibition or blocking, often with complete mutism which may appear as an initial symptom but may later be partially overcome with a return of speech that is distorted; or speech may gradually 'deteriorate' or 'regress' by use of

simpler language forms, dropping out of connecting words, loss of more recently acquired language forms, and fragmentary speech. Language may be used as a repetitive expression of anxiety with questions concerning identity and orientation, but never waiting or attending to an answer or ceasing to use it as a means of communication or interpersonal relationship; it is used as a chanting accompaniment of motor play. There are occasional explosive expletives, aggressive or obscene in nature."

According to these descriptions of children's use of language, no child would be entirely safe from the diagnosis of schizophrenia. Other characteristics, which develop into a self-contradictory syndrome are described. "Growth discrepancies are marked. The children are too big or too little, too fat or too thin . . . the physiological rhythms of daily living lose their normal rhythmic pattern. This is seen in the sleeping, eating, and elimination habits. Mothers complain of it as a part of the early disorder in behavior, and we observe it in trying to adapt the children to the ward routine suitable for the normal child. . . . Characteristic disturbances in patterned motor behavior or motility can be demonstrated in every schizophrenic child. Mothers will tell us in retrospect of unevenness in the motor development of the child. They complain of motor awkwardness and poor control of limbs. . . . There are postural reflex responses which are nearly specific for childhood schizophrenia. Postural reflexes are tested by having the child stand with his arms outstretched in front of him and his eyes closed while the head is turned on the neck by the examiner. The primitive response to this test is to turn the body so as to bring it in line again with the head. . . . The schizophrenic child responds with a graceful fluid whirling which he quickly accepts as a new pattern of activity, if he has not already discovered it himself, and will carry it on spontaneously. Sometimes the examiner need only put his finger on the top of the head which suggests a pivot."

How then are we to reconcile "motor awkwardness and poor control of limbs" with "graceful fluid whirling" and "when left to his own devices, he carries on endless rhythmic and graceful dancing behavior with changing tempos"? "The schizophrenic child shows a physical or bodily dependence which can be seen by simple physical contact. He seeks instantly to use the body or the motility of the adult and will either lean so completely on the other

person as to fall to the ground if that support is withdrawn or will readily sink completely and passively into the lap and arms of the accepting adult as though to melt his very body into that of the other and thereby identify himself with the other's more secure center of gravity. With the same impulse, he clings to and holds on to the adult at every opportunity."

Any parent who has not had this experience with a child is a rare exception. It becomes increasingly difficult to delineate normal behavior for a child. They must (literally and figuratively) not know which way to turn.

Other psychiatrists present varying criteria for diagnosis. Bradley[7] notes eight behavior characteristics: ". . . seclusiveness; irritability when seclusiveness is disturbed; daydreaming; bizarre behavior; diminution of personal interests; regressive nature of personal interests; sensitivity to comment and criticism; physical inactivity." He concludes that ". . . since schizophrenia appears to be a *way of life* (italics mine) rather than a disease, its existence is not incompatable with that of other handicaps such as mental deficiency, epilepsy, or cerebral palsy, to mention only a few coexisting conditions that have come under the writer's observation."

In pediatrics should one treat a "way of life" with electric shocks? Despert[14], who has investigated and written extensively on childhood schizophrenia, states: "There is actually no satisfactory formulation of childhood schizophrenia as a disease entity; the symptomatology, on the whole, is described by comparison with the adult syndrome, or in terms of its later developments, during adolescence and adulthood." She lists these characteristics: "Dissociative and autistic manifestations; excessive dependence on mother; bizarre behavior such as motor rigidity, nonfunctional play, rituals and compulsions; temper tantrums; speech and language deviations, hallucinations, delusions."

This formulation of symptomatology in comparison with the adult syndrome leads to gross scientific inaccuracies. One reads, for instance, that "one may find schizophrenic symptoms in cases of hysteria." That is like saying one may find symptoms of tuberculosis in the common cold. It is an adjective charged with serious connotations and should not be applied loosely or with lack of clarity.

Since the diagnostic criteria of this entity have produced such

diversity of opinions, it would be indeed helpful if there were some long term study of this condition, let us say, a follow-up study of children diagnosed as psychotic or schizophrenic for one or two decades to determine whether they ever developed the clinical picture of adult schizophrenia and psychosis. Unfortunately, only one such study exists. Sherman and Jost[35] report a study of 35 children whose condition was diagnosed as psychotic and who were observed for periods ranging from only two to ten years. A detailed history of the behavioral development of each child was obtained from parents and teachers. Ten of the 35 were committed to state hospitals for the insane by the time they were twenty. And the conclusions are astonishing. "The majority of the 35 patients were intellectually normal during their infancy and early childhood and no one in the family suspected at that time that they were different from other children. In 26 cases the mental abnormalities developed in a recognizable form when they were between eleven and thirteen." In all but one case the parents stated that the behavior of the child was good before he manifested the nervous or other abberant activities. *"Indeed, the majority of the subjects were model, well behaved children, who gave their parents and teachers no trouble"* (italics mine).

A similar observation of the childhood behavior of psychotic adults was made by Bowman[6]. His survey of 322 cases, the data provided by parents, friends, teachers, etc., shows that in ". . . schizophrenia there are more model children . . . they tend to have few friends, to indulge in solitary amusements, to be followers, feel superior, to be closemouthed and uncommunicative. . . . As a child, he is not oversensitive and daydreams less than normal."

Further confirmatory evidence was produced by Buren[10] in a study of psychological examination of children who later became psychotic. He concludes that "children who will later develop a mental disease of sufficient seriousness to require hospitalization do not differ greatly as a group from other children at the school ages when they were examined by the Bureau of Child Study. . . . Within the group of prepsychotic cases, however, the children who later became schizophrenic tend to be apathetic in the childhood examining situation and are also more intelligent when compared to the constitutional mental disease group, who are excitable and of lower intelligence."

A study of the mothers of schizophrenic patients (adults) by Tietze[57] is very revealing. When asked about the prepsychotic personality of their schizophrenic children, a characteristic first response was: "There was nothing wrong with him; he was the best child, he worshipped me, he was the least trouble of all my children, the easiest to be trained; as far as I could see he was perfectly normal." The children were shy and timid, especially with persons outside the family circle. They were reluctant to mingle with other children and if they did it was at the initiative of their playmates or mothers. In a play situation they were followers rather than leaders, unaggressive, poor losers and unable to take being teased. Only 6 (out of 25) reacted to frustration with temper tantrums. The other 19 never have shown signs of a temper. The mothers' description of the prepsychotic personality of their children shows how highly they prize "normality" and "goodness" and how inseparable these two concepts have become in their minds. These cases became openly psychotic in college. Previous to that, they showed only poor social adjustment.

It becomes increasingly apparent that only in reasoning backwards, and investigating the history *after* an adult psychosis has developed that a clear picture of the prepsychotic personality in children is revealed. These studies show a remarkable unanimity of opinion, completely contradicting Bender's elaborate symptomatology. However, there is even danger here, for, in the diagnosis of problem children, it is apparently easy to be misled into generalizations about diagnosis on the basis of this picture of the prepsychotic personality.

Kemble[37] warned against this easy pitfall. "There can be no doubt that some individuals with withdrawn personalities will become schizophrenic, but this is quite different from saying that all withdrawn types are potential schizophrenics."

Examination of the results of shock treatment is described by Des Lauriers and Halpern[13] who tested one hundred children from four to eleven years of age, before and after shock therapy. These are their conclusions: "On the basis of our experience it can be said that no one interest pattern can be considered significant or diagnostic of childhood schizophrenia." Again, "in attempting to summarize the overall effect of shock treatment on the schizophrenic child, as it shows itself through the medium of the various

tests used, the chief impression obtained is that the shock treatment has broken down all the superadded structures, the neurotic and anxious features, and there now appears in strong relief a clearly defined picture of schizophrenia."

Unfortunately this "clearly defined picture" is never defined, nor clear. "There is no improvement in the mental continuum, there is no change in reasoning and judgment, the tendency to accept incidental and far-fetched issues as important is still present. On the projective tests, the main change is a general flattening out of the affect. . . . The Rorschach records therefore appear to be all more or less of one pattern, a schizophrenic pattern, with definitely fewer neurotic attributes than existed in the preshock protocol. *If shock therapy did nothing else, it served to clarify diagnosis*" (italics mine).

Ordinarily, in the process of scientific medicine, there is a logical progression of investigation and treatment for most diseases. New drugs are discovered and theories of action are formulated. These new drugs are discarded or used and conclusions of efficacy drawn from them. Physicians hesitate to use a topical sulfa preparation for fear of sensitizing a child to the drug and perhaps precluding its oral use in a more serious illness. Hormone injections are followed closely for untoward reactions and serious complications. Immunizing sera are carefully tested to prevent anaphylactic reactions; recurrences of febrile convulsions prevented. Language is edited before parents, and concern dissembled to prevent unnecessary alarm. And yet—before the effects of shock treatment on children have been properly evaluated, there are on record eleven cases of prefrontal lobotomy performed on children.[18]

Freeman and Watts[18] in their article claim that the outstanding feature of childhood schizophrenia is seclusiveness . . . and that ". . . children who are to develop schizophrenia before puberty may show this seclusiveness before the age of two. Indeed, one patient is reported to have turned definitely away from other children at the age of nine months." They claim that incongruity of affect has been frequently noted, and as an example of this cite one patient who ". . . handled snakes and worms without concern while at the same time she expressed ungovernable alarm about crossing a bridge."

One should at least be fair to children and understand them.

This child is afraid to cross a bridge, but is not afraid of handling snakes and worms. To conclude "incongruity of affect" is a misapplication. It too obviously shows the subjectivity of diagnosis and the attempt to force a simple observation into the symptom of psychosis. There is more evidence of the assumptive technique of diagnosis. It is stated that "hallucinatory and delusional experiences are not so readily brought out in these children as they are among older schizophrenics, but they probably occur quite frequently. The reason that they are not more frequently reported is due to the stubborn desire of the child to keep his fantasy-life undisturbed."[18]

The purpose of prefrontal lobotomy for the schizophrenias occurring in childhood has been undertaken ". . . with a view to terminating the fantasy-life and reducing the expenditure of emotional energy upon the self. . . . It is easier to smash the world of fantasy, to cut down upon the emotional interest that the child pays to his inner experiences, than it is to redirect his behavior into socially acceptable channels."[18]

It becomes necessary again to come to the defense of children and their fantasy-life. Clinical tests[1] reveal that the mental life of adult schizophrenics is comparable to the fantasy-life of a child. But children normally live in a world of fantasy and imagination and in later life many of their artistic accomplishments in fields of literature, art and professions are directly derived from this. It would seem reasonable to permit them at least the opportunity for developing a sense of reality before destroying their potentialities.

In the technique of prefrontal lobotomies a great deal of frontal lobe tissue has to be sacrificed in children in order to obtain any more than "temporary improvement." The younger the age at which the psychosis has begun the more posteriorly the incisions have to be made, with resulting greater disability, it is claimed. The youngest patient in this series of eleven cases was four years and six months at the time of operation. "Absolutely incorrigible, destructive, assaulting, his face was a mass of bruises as the result of banging his face against furniture in response to the most trivial frustration. . . . Unfortunately the possibilities in this case will remain unknown because after return home, and when things were going well, he contracted meningitis and died two weeks after operation."[18]

There is another rationale expressed by the authors for continuing to perform this operation. "If fear and hate can be eliminated by operation in adults and render it easier for them to get along in their social milieu, then prefrontal lobotomy in children could conceivably enable them to develop their remaining resources to a degree adequate for constructive living."[13]

Apart from the questionable rationale of eliminating fear and hate in human beings, what are the other, concomitant functions which are eliminated by lobotomy?

Psychological testing has revealed specific eliminations of cerebral functions[17, 25, 30, 32, 46, 51, 53]. There is a reduced capacity for prolonged attention and effective planning; a lack of normal degree of deliberative qualities and the patients exhibit defects when presented with tasks which require analysis and synthesis. There is a lack of self-consciousness, restraint, embarrassment and an indifference to criticism. They tend to be tactless, smug, self-satisfied. In extreme cases, they are indolent, lacking in ambition and careless in personal tidiness. Frankly abnormal ideational features, such as delusions and hallucinations, may not disappear but the emotional reaction to these disturbing experiences is dulled. There is an incidence of 12.3 per cent in whom convulsions occur, even as late as nine to twelve months after treatment. It causes loss of the power of abstract thinking.

From the pediatrician's point of view one may be a little concerned over the prognosis which these new procedures present. *Primun non nocere!* Above all do no harm.

BIBLIOGRAPHY

1. Abt and Bellak: Handbook of Projective Psychology (in press).
2. Aldrich, C. Anderson: The Pediatrician Looks at Personality. Am. J. Orthopsychiat., 17: 571, 1947.
3. Bender, Lauretta: Clinical Study of 100 Children. Am. J. Orthopsychiat., 17: 40, Jan. 1947.
4. Black, H. R.: Smith, O. C. and Bruch, H.: Schizophrenia in a Four-Year-Old Boy. Am. J. Psychiat., 100: 805-810, May 1944.
5. Bosselman, B.: Evolution of a Schizophrenic Process in a Young Boy. Psychiatric Quart., 19: 258-266, April 1939.
6. Bowman, Karl: A Study of the Pre-psychotic Personality in Certain Psychoses. Proc. A. Research Nerv. & Ment. Dis., 180-210, 1933.
7. Oltman, Jane E.: Psychosurgery—A Review. J. Am. M. Women's A., May 1950.
8. Bradley, Charles: Biography of a Schizophrenic Child. Nerv. Child, No. 1 & 3, 1942.
9. Bradley, C.: Schizophrenia in Childhood, 1941.
10. Buren, James E.: Psychological Examinations in Children Who Later Became Psychotic. J. Abnor. & Social Psychol., 39: 84-96, Jan. 1944.
11. Cottington, Frances: Treatment of Childhood Schizophrenia by Metrazol Shock. Am. J. Psychiat., 98: 397, 1941.
12. Creak, M.: Psychoses in Children. Proc. Roy. Soc. Med., 31: 519, March 1938.
13. Des Lauriers and Halpern, F.: Psychological Testing in Schizophrenia. Am. J. Orthopsychiat., 17: 57, Jan. 1947.

14. Despert, J. Louise: The Early Recognition of Childhood Schizophrenia. M. Cin. North America, 31: 680-687, May 1947.
15. Dittmer, Thomas: Juvenile Schizophrenia. Quart. Bull. Indiana Univ. Med. Center, 5: 8-10, Jan. 1932.
16. Eickhoff, L.: Acute Schizophrenia in Childhood—Case Study. Edinburgh M. J., 51: 201-204, April 1944.
17. Frankl, L. and Mayer-Gross, W.: Personality Change After Prefrontal Leucotomy. Lancet, 2: 830-824, Dec. 1947.
18. Freeman and Watts: Prefrontal Lobotomy in Children—11 Cases. Digest Neurol. & Psychiat. Inst. of Living, 202-220, April 1947.
19. Freeman and Watts: Intelligence Following Prefrontal Lobotomy in Obsessive Tension States. J. Neurosurg., 1: 291, July 1944.
20. Freeman and Watts: The Surgical Treatment of Mental Disorders. Med. Ann. Dist. Columbia, 8: 35, Dec. 1939.
21. Freeman and Watts: The Radical Treatment of the Psychoses and Neuroses. Dis. Nerv. System, 3: 6, Jan. 1942.
22. Freeman and Watts: Prefrontal Lobotomy—An Evaluation of 200 Cases over 7 years. J. Ment. Sc., 90: 532, April 1944.
23. Freeman and Watts: Prefrontal Lobotomy—Survey of 331 Cases. Am. J. Ment. Sc., 211: 1, Jan. 1946.
24. Freud, Anna: Psychoanalysis in Children, 1946.
25. Friedlander, Joseph W. and Banay, Ralph S.: Psychosis Following Lobotomy in a Case of Sexual Psychopathy. Arch. Neurol. & Psychiat., 59: 302-311, March 1948.
26. Garieso, Aquiles and Sagreras, Pedro O.: Shock Cardiazolico en la Infancia. Arch. Argent. de pediat., 21: 7, Jan. 1944.
27. Garmany, Gerald: Schizophrenia in a Boy of Eleven Years. J. Ment. Sc., 93: 772-777, Oct. 1947.
28. Gladstern, H.: The Later Adjustment of Twenty-Six Adolescents Diagnosed as Schizophrenic or Potentially Schizophrenic. Smith College Studies Social Work, 2: 106-136, 1941.
29. Goldensohn, L. N.; Clardy, Ed. R. and Levine, Kate N.: Schizophrenic-like Reactions in Children. Psychiatric Quart., 19: 592-604, Oct. 1945.
30. Halstead, W. C.; Carmichael, H. T. and Bucy, P. C.: Prefrontal Lobotomy. Am. J. Psychiat., 103: 217, Sept. 1946.
31. Hartwell, S. W.: Study of Twenty-Five Children Presenting Withdrawal Type of Personality. Am. J. Orthopsychiat., 2: 143-151, 1932.
32. Heilbrunn, G. and Hletko, P.: Disappointing Results With Bilateral Prefrontal Lobotomy in Chronic Schizophrenia. Am. J. Psychiat., 99: 569, 1943.
33. Jenkins, R. L. and Glickman: The Schizoid Child. Am. J. Orthopsychiat., 16: 255-261, April 1946.
34. Kanner, Leo: Autistic Disturbances of Affective Contact. Nerv. Child, 2: 217, 1942.
35. Kanner, Leo: Child Psychiatry, 1932.
36. Keiser, S.: Severe Reactive States and Schizophrenia in Adolescent Females. Nerv. Child, 4: 17-25, Oct. 1944.
37. Kemble, R. P.: Adolescent Pre-psychotic Conditions—Criticism of a Concept. Smith College Studies Social Work, 2: 97-105, 1941.
38. Klein, Melanie: The Psychoanalysis of Children, 1932.
39. Knight, Robert P.: Adolescent Catatonic Schizophrenia With Mutism. Psychiatry, 9: 323-339, Nov. 1946.
40. Leitch, Mary and Schafer, S.: A Study of Thematic Apperception Tests of Psychotic Children. Am. J. Orthopsychiat., 17: 337, 1947.
41. Lorand, S.: The Psychoanalytic Contribution to Treatment of Behavior Problems in Children. Am. J. Psychiat., 105: 357-360, Nov. 1948.
42. Lourie, R. S.: Pacella, B. L. and Piotrowski, Z. A.: Prognosis On Schizophrenic-like Psychoses in Children. Am. J. Psychiat., 99: 542, 1943.
43. Lurie, E. B. and Tietz, Hertzman, J.: Functional Psychoses in Children. Am. J. Psychiat., 92: 1169, March 1936.
44. Mahler, M. S.; Ross, John R., Jr., and DeFries, Zira: Clinical Studies in Benign and Malignant Cases of Childhood Psychosis. (Schizophrenic-like). Am. J. Orthopsychiat., 19: 295, April 1949.
45. Odriosola, Ricardo: Success in Electroshock Therapy in a Three-Year-Old Girl. Arch. de pediat. d. Uruguay, 17: 385, July 1946.
46. Oltman, Jane E.: Psychosurgery—A Review. J. Am. Med. Women's A., May 1950.
47. Peck, H. B.: Rabinovitch, R. and Cramer, Joseph B.: A Treatment Program for Parents of Schizophrenic Children. Am. J. Orthopsychiat., 19: 592, Oct. 1949.
48. Polaten, P.; Friedman, M. M.; Harris, M. M. and Horowitz, W. A.: Vertebral Fractures Produced by Metrazol Induced Convulsions. J. A. M. A., 112: 1685, April 1939.
49. Potter, A. W. and Klein, H. R.: Evaluation of Therapy of Problem Children as Determined by Follow-up Study. Am. J. Psychiat., 94: 681, Nov. 1937.
50. Reitman, F.: Post-operative Lobotomy Personality Changes. J. Nerv. & Ment. Dis., 105: 532, June 1947.
51. Robinson, Mary F.: What Price Lobotomy? J. Abnormal & Social Psychol., 41: 421, Oct. 1946.
52. Rothschild, D. and Kaye, A.: Effects of Prefrontal Lobotomy on the Symptoms of Dementia Precox—100 Cases. Am. J. Psychiat., 105: 752-759, April 1949.

53. Russell, Ritchie W.: Functions of the Frontal Lobes. Lancet, 1: 356-360, March 6, 1948.
54. Schumacher, Henry C.: Schizophrenia in Infants and Children. Ohio State M. J., 42: 1248, Dec. 1946.
55. Sherman, Mandell and Jost, Hudson: Diagnosis of Juvenile Psychosis. Am. J. Dis. Child., 65: 868-872, June 1943.
56. Spock, Benjamin: Preventing Early Problems. Am. J. Orthropsychiat., 17: 575, 1947.
57. Tietze, Trude: A Study of Mothers of Schizophrenic Patients. Psychiatry, 12: 55-65, Feb. 1949.
58. Wechsler, I. S.: A Textbook of Neurology, 1940.
59. Wertham, F. and F.: The Brain as an Organ, 1934.
60. Wertham, Frederic: Dark Legend, 1941.
61. White, M. A.: A Study of Schizophrenic Language. J. Abnormal & Social Psychol., 44: 61-74, Jan. 1949.

HEREDITY OF DIABETES MELLITUS. (Medizinische Klinik. Munich, 46: 327, March 16, 1951). The question whether it is advisable for diabetic patients to marry induced Thöne to investigate the familial occurrence of diabetes mellitus in a group of 130 diabetic patients. Forty-one (31.5%) of these patients had persons with diabetes mellitus among their blood relatives. In 15 of these patients one of the parents was diabetic (nine mothers and six fathers); in three, one of the parents as well as siblings had diabetes; in six, one of the grandparents was diabetic, whereas the parents were not; in about one-fourth of the patients siblings were diabetic, and conjugal diabetes was found in two couples. When diabetes was transmitted from parents to child, it appeared at an average age of 52, whereas when a grandparent was diabetic it became manifest at an average age of 36. Patients who have diabetic siblings usually become diabetic at about the age of 46. Anteposition was quite evident; that is, even if diabetes appeared at an advanced age in the older generation it was likely to appear earlier in the later generation. A predisposition to diabetes is hereditary, but the transmission is oftener recessive than dominant. Although the likelihood of developing the disease is much greater for a child with a familial predisposition to diabetes than for one without such predisposition, the author does not believe that a diabetic person should be forbidden to marry, at any rate, not if the partner is free from metabolic disorders. If both partners have a family history of diabetes, then the likelihood of diabetes in the offspring is great.—*Journal A.M.A.*

CLINICAL REVIEW

In order to encourage the writing of clinical articles by recent graduates or senior medical students, the ARCHIVES *will publish monthly at least one such paper from the classes of Doctor Reuel A. Benson, New York Medical College, New York, and Doctor Philip Moen Stimson, Cornell Medical School, New York. Other interested medical schools are cordially invited to submit student papers for consideration.*

EPIDEMIC DIARRHEA OF THE NEWBORN*

J. DOUGLAS HALLOCK, M.D.
New York.

In 1937 Rice and Best[1] first called attention to an epidemic form of diarrhea which occurred at random among babies in hospitals. Reporting on eleven New York City institutions, they revealed an incidence of 505 cases out of 3,672 live births during the period 1934-36. Most affected was the newborn, during the first to third weeks of life. Etiological factors were entirely unknown, nor was there any apparent relation to sex, race, season, nutritional state or to feeding problems.

Usually the first indication of trouble was a cessation of weight gain, listlessness and refusal of food. Subsequent symptoms were those of acute intestinal toxicosis with the development of yellow or yellow-green watery stools. Significant pathological findings were consistently absent. There was congestion of superficial intestinal vessels, mesenteric lymph node enlargement and congestion of Peyer's patches in the ileum.

It is now realized that this epidemic form of diarrhea in the newborn is not a new clinical entity[2] but is only one of many diarrheal disorders. The modern era of hospital deliveries has served to reduce maternal mortality by effecting deliveries where facilities are present to handle most of the accidents of childbirth. On the other hand,[3] by bringing a large group of babies together, all may be exposed by one individual who harbors an agent capable of producing disease. Epidemic diarrhea, therefore, attains prominence as a result of modern custom, since it does not occur as an epidemic among home-born babies. By way of example,[15] the Boston Lying-In Out-Patient Department reported 1,015 home deliveries for the year 1935, as compared to 47 in 1945 and none

*From the Department of Pediatrics, Cornell University Medical College, New York.

in 1946. Although maternal mortality has decreased through the years, the question resolves, "May not the home be safer than the hospital for the newborn, in certain respects?"

In the United States, infant morality[2] did not decline until 1910. At that time it was 135 per 1,000 live births. During 1949, 32 died per 1,000 births. A significant portion of this disease is due to the lowered incidence of diarrheal diseases, especially among those over one month of age. Among the neonatal group the improvement has been slight, so that 30.4 per cent of diarrheal deaths still occur in those under one month of age. A breakdown of mortality figures[4] for the years 1923-47 reveals the following: Infant mortality of those one month to one year fell from 34.5 to 6.2 per 1,000 live births, while in those under one month it fell only from 31.9 to 20.2 per 1,000 live births. The lag in the neonatal group is chiefly due to congenital malformations, prematurity and birth injuries. The diarrhea death rate has fallen from 8.6 to 0.9 in the group one month to one year, while in those under one month, it has only decreased from 1.2 to 0.5 per 1,000 live births. This is due largely to an epidemic form of diarrhea of unknown etiology. Among prematures, this form of diarrhea constitutes a great disaster, while of negligible mortality in those over six months. In New York City alone, during the period 1934-48, there were 184 outbreaks of epidemic diarrhea in which 2,966 of 18,542 newborn were attacked, a morbidity of 16 per cent. In three of these years diarrheal deaths were greater than the total due to typhoid, scarlet fever, measles and whooping cough. Nine hundred twenty-four died (5 per cent), or a case fatality of 31.2 per cent. Others[5, 6] have collected cases whose fatality rates varied from 10.5 to 81.8 per cent.

It is a well-known fact that an epidemic may occur in any hospital, no matter how well operated and despite ordinary precautionary measures. The most insidious nature of this process is that almost epidemic proportions have developed before it is recognized that something is amiss. During late August 1944, two infants in separate nurseries of Temple University Hospital[5, 7] developed loose stools. One lost weight, was immediately isolated and recovered uneventfully. The second developed no further symptoms, was not isolated until the second day, and also recovered. By September 1, a full blown epidemic had developed. On

successive days others in one nursery developed diarrhea and one previous discharge was readmitted with vomiting and weight loss. Nine days after the initial episode, two babies in the premature unit were found to have loose stools. This group had separate quarters and separate nursing personnel. All infants with diarrhea were placed in the regular pediatric ward with separate nursing facilities and personnel for the remainder of the epidemic. While there, none of the other children, from one to eighteen months, developed symptoms as a result of these contacts. The nurseries were closed four days and reopened, but mild cases continued to recur every few days. Finally, in order to eradicate the epidemic the nurseries were again closed, cleansed and repainted. Of 346 patients during this period, 63 developed diarrhea (18.2 per cent morbidity) with a case fatality of 23.8 per cent. Although there was great variety in the severity of symptoms, the prematures were more severely affected and suffered the higher fatality rate. Cultures from patients, personnel, and rooms failed to reveal any known pathogenic organism, nor was any break in technique discovered.

Foster[8] relates in detail the difficulties in confining an outbreak, even under an ideal situation.

"An outbreak of epidemic diarrhea of the newborn at the Cambridge City Hospital originated with the admission of two babies who developed diarrhea at home and were admitted to the pediatric ward in the main building on December 9 and 11, respectively. The admission diagnosis in the first case was gastroenteritis, and enterocolitis in the second case. Both babies died—the first 22 days after the onset of the disease, the other on the ninth day.

"On December 11, two days after the admission of the first case from the outside, two additional babies in the pediatric ward developed diarrhea. On December 13, there were two more cases. Then, after a quiet interval of six days, two cases followed on December 19, two more on December 27 and one on December 28.

"The number of infants in the pediatric ward who developed diarrhea totaled thirteen. All were under ten months of age. Older children, who were as intimately exposed as these infants, escaped infection. The clinical picture was that of listlessness, inappetence with resulting cessation of gain or loss in weight, fol-

lowed by copious, watery yellow or green stools with or without vomiting or fever, usually more or less distention, and, in severe or fatal cases, dehydration, ashen grey pallor, circles beneath the eyes and depression of the fontanelle. Convulsions occurred in one case just prior to exitus. Stool specimens submitted in each case to the state laboratory were uniformly reported negative for organisms of the dysentery and salmonella groups.

"The incubitory period appeared to vary from two to fourteen days. The average duration of the disease was six days, with individual variations from two to 22 days. There were four deaths in the group—a mortality of 31 per cent. The morbidity rate was 43 per cent.

"The first two babies, who developed diarrhea in their homes prior to admission, as well as all subsequent contact cases, were isolated on one side of the pediatric ward with individual screening. Admissions to this ward were interdicted and all babies, excepting diarrhea cases, were subsequently admitted to an auxilliary pediatric ward opened on the second floor of the main building. A barrier was declared in the center of the pediatric ward, and separate staffs of nurses were assigned to the care of diarrhea cases on one side and noninfected cases on the other. Rigid nursing techniques were enforced with special emphasis on individual thermometers, hand-scrubbing, and the use of gowns, masks and head gear.

"Upon the occurrence of the first contact cases, suspicion arose that we were dealing with the so-called epidemic diarrhea of the newborn, and our fears were reported to the District Health Officer by telephone. Suspicion became conviction when negative laboratory reports began coming in and the first deaths occurred on December 18, nine days after the admission of the first case.

"There was great apprehension that the infection might spread to the nurseries in the maternity department although it was housed in Cahill House, a separate building. All formulas for the pediatric ward, formerly prepared in the Cahill House formula room, were prepared in the diet therapy room used for teaching purposes, in the nurses' home. There was no connection with Cahill House, either by resident physicians, nurses, housekeeping personnel or others—both the main house and Cahill House having separate staffs. The only medical personnel who saw babies in

both the pediatric ward in the main house and in the nurseries in Cahill House were pediatric consultants and visiting staff pediatricians. All other possible avenues of transmission from the pediatric ward to the nurseries were believed to be blocked.

"Despite these precautions, four babies in the premature nursery on the third floor of Cahill House developed diarrhea on December 27. One of these was signed out against advice and the other three were isolated immediately in a separate nursery with a separate staff of nurses assigned to their care. On December 28 a fifth case occurred among the prematures, and it was isolated in a separate room, off the infected nursery. Additional cases among the prematures occurred on December 29 and 30. The third floor of Cahill House was closed and admissions were limited to the first and second floors.

"No new cases occurred for fifteen days, then, on January 14, a suspected case of epidemic diarrhea appeared in one of the nurseries on the first floor of Cahill House; and upon the appearance of another case the following day, the decision was made to close the maternity service. The outbreak was limited to nine infants—seven of whom were prematures. The duration of the disease varied from six to 28 days. The average duration was ten days. There were four deaths in this group—all among prematures. The mortality rate was 44 per cent.

"With the closure of Cahill House, the second phase of the epidemic terminated; but the Cahill House episode did not end there." Thirteen babies who were born in Cahill House during the epidemic, and were free from signs or symptoms of infection when discharged on the seventh day after birth, subsequently developed diarrhea in their homes. Seven of these cases were readmitted to the Cambridge City Hospital, and six entered other institutions. The period elapsing between the dates of discharge and onset of diarrhea ranged from one to 31 days. The average period was eleven days. Duration of the disease was from four to 26 days; with an average of ten days. There were no deaths in this group. In three of the cases in this group periods of 28, 29 and 31 days, respectively, elapsed between discharge and the onset of diarrhea. As 21 days is usually viewed as the maximum period of incubation, it is questionable whether these infections were contracted in Cahill House or subsequently at home.

"With readmission of the last case attributable to Cahill House on January 24, the epidemic ended. Recapitulation shows that 35 cases of infectious diarrhea occurred during the three phases of the epidemic. The mortality—with eight deaths—was 22.4 per cent."

The earliest consistent sign of this malady is the loss of appetite with an arrest of weight gain[3, 4]. In 48 hours the toxic phase begins with frequent, expulsive, watery stools of green (if breast fed) or yellow-brown color, but without blood or pus. The temperature rises and remains about 99°-100° F., seldom above 101° F. Dehydration follows with subsequent acidosis, and vasomotor collapse if unchecked. Occasionally there is a slight amount of redness to the tongue margin. St. Gene[13] has claimed that low CO_2 is frequently a prodrome of infectious diarrhea[14], and feels that the primary infection frequently becomes insignificant when acid-base balance is restored.

During the invasive state the process may be confused with intestinal disturbances due to overfeeding, improperly adjusted formula preparations, cathartics or drugs. However, its specificity for the newborn without spread to older children and adults, the lack of high initial temperature rise with blood or mucus in the stools, do not tend to confuse it with salmonella or typhoid. In this regard, the agglutinin titer and microscopic examinations prove fruitless. Laboratory findings include an increase in total protein, nonprotein nitrogen, and chloride, with a marked decrease of CO_2 combining power as low as 5-10 volumes per cent. Studies have shown, however, that the newborn normally has a lower CO_2 and higher chloride value[9]. Complications include otitis media, bronchopneumonia, liver parenchymal degeneration, dural vessel congestion and, occasionally, renal thrombosis[10].

There is a remarkable lack of post-mortem pathological findings in comparison to the severity of the symptoms. The intestinal mucosa is usually hyperemic and atonic[11] but without hemorrhage or ulceration, as a rule. Shallow ulcers[11, 12] and denuded mucosa have been found at times, however. Brain edema[11], with extravasations and diapedesis, have also been noted. As previously stated, there is no relation to sex, race, nutritional state (unless debilitated) or season[1, 4]. The disease is most common in the north temperate zone.

The median postnatal age is nine days[4] with a median incubation

of seven days[2, 4, 6, 14]. Minimum incubation is considered to be one day and the maximum, by most, to be 21 days[8, 14]. Others feel the incubation may last as long as 31 days[4, 6]. Fatal processes last anywhere from one to 25 days (median seven days) while in those who recover it may last from three to 26 days (median twelve days).

Attempts to incriminate a single etiological agent have so far been uniformly unsuccessful. In reviewing those epidemics in which adequate epidemiological studies were conducted, it would seem that the cause of this epidemic form of the newborn disease is a miscellaneous etiological group bound together by diarrhea[15]. At one time or another, suspicion has been cast upon bacteria, both pathogenic and so-called nonpathogenic groups, as well as a possible viral source. One factor is apparent, however, the appearance of an epidemic is not merely chance but due to breaks in hospital technique, some due to gross carelessness, others relatively minute. Crowding of patients, insufficient number of personnel are the most common precipitating factors[3]. Clifford[15] reported five epidemics, in each of which it was later learned that either a mother or a nurse had developed diarrhea prior to the epidemic. Later investigations found the community milk supply to be grossly contaminated with B. pyocyaneus. Escherichia coli[16] was considered the agent in four epidemics. Hospital A had 62 infants, of which 26 became ill, with three subsequently dying. Hemolytic E. coli was found in fourteen of sixteen stools and in the nursing supervisor's stools. A later epidemic in the same hospital affected 31 infants, with seven deaths. The same organism was found in stools of seven of eight infants, four of seventeen nurses, and in the milk in high numbers. In the other hospitals, E. coli was found in a significant percentage of the stools belonging to the nursing personnel, in the evaporated milk and on the rubber nipples.

One New York hospital experienced three outbreaks[17]. In the first, stool examinations revealed E. coli mutabile, alpha hemolytic streptococcus aureus, staphylococcus aureus, stap. albus B. subtilis, hemolytic E. coli, aerobacter aerogenes, none of which were consistent enough to be significant. The second attack revealed significant findings of E. coli mutabile and hemolytic E. coli in thirteen prematures. The third outbreak failed to reveal any suspicious organism. B. dispar[8] was found present in significant

amounts in 36 infants during a Baltimore epidemic, 21 of whom died. Paracolon bacilli and pseudomonas have also been found[19].

Unfortunately, most epidemics shed no light as to the causative agent but do reveal faulty technique due to crowding and insufficient medical staffs[20-23]. In one case seven nurses used a common rectal thermometer without adequate sterilization. A bacterial count revealed seven million viable bacteria, mostly E. coli. In another case, "sterilizing solutions" had 75 to 150 thousand organisms per cubic centimeter, including S. aureus, E. coli, alpha Streptococci, Lancefield group D, and B. subtilis. In still another, the formula samples and nipples revealed 75 to 396 thousand bacteria per cubic centimeter. It is not unusual to learn of babies being diapered prior to feeding, hands washed in tepid water with little soap, rubber nipples boiled less than twenty minutes and floating on the surface, and improper cleansing of maternal breasts.

Several investigators[4, 24, 25] have suggested that this condition may result from the continued change in the nasopharyngeal and intestinal flora of the newborn during the early weeks of life. This in turn may cause metabolic disturbances. The lack of proper flora may be due to conditions of sterile delivery and subsequent nursing care. Breast fed children[26] develop mostly Gram-positive intestinal organisms with a predominance of B. bifidus-acidophilus. There are relatively few colon bacilli. Artificially fed infants develop a mixed intestinal flora with a predominance of B. coli. Strep. anhemolyticus, B. bulgaris, B. proteus, and diphtheroids are also found. The feeding of 12 per cent lactose to an artificially fed child leads to reversion of the flora to the breast fed type. Investigation of an epidemic of seventeen cases in Brooklyn Methodist Hospital[25] revealed that only one baby of the nine who died had been breast fed (and only for four days) while the remainder had been exclusively fed by the bottle. Only four breast fed infants had been affected at all. Stool cultures of eight affected patients revealed streptococci in seven, staphylococci in four, B. coli in five. Neither strep. nor staphylococci are found in normal infant flora and may have resulted from the failure to develop a normal flora. As a result, it may be that these foreign organisms could not be combatted properly. It is said that vaginal antiseptics[25] rob the child of the bacteria which he normally collects during the birth process. Meconium, which is supposed to contain organisms

essential to digestion, is found to lack the normal Gram-negative bacteria under such conditions. In fact, most are sterile. It is also claimed that prior to the use of vaginal antiseptics at Methodist Hospital there were no epidemics of newborn diarrhea.

Attention has been directed to the fact that hospital epidemics of diarrhea also occur concurrently with community outbreaks of influenza[11] and gastric flu[27]. In attempting to ascertain a possible virus etiology, Buddingh[28] has devised a method of inoculating stool filtrates (Berkfeld V candles) into the scarified corneas of rabbits. This produces what he considers a characteristic change of hyperemia and swelling of the palpebral conjunctiva and cloudiness in the scarified area within 24 hours. After 48 hours the process rapidly disappears leaving only haziness in the lines of deeper scarification. Microscopic sections show focal neutrophilic accumulations around the area of necrosis, and epithelial ulceration in the area of scarification. Intracytoplasmic and intranuclear inclusion bodies have not been found to date. However, control material from the throats and stools of normal individuals indicate that the reaction is specific. Buddingh was also able to obtain complete immunization to eye re-inoculations in about three weeks. The virus could also be transferred to another eye by corneal extracts. Attempts to propagate the agent in guinea pig and rabbit skin and in the chick embryo were without success.

Unfortunately, he does not describe in detail the difference in eye reactions between those from diarrheal patients and his controls. The main objection[15] seems to be that others have obtained the same reaction by merely scarifying the cornea without inoculation. Cummings[30] used material from two epidemics with negative results in all cases. His serial sections revealed the nonspecific reaction due to irritation. Controls with trauma, Monilia, staph. and streptococcal strains, in addition to washings of normal throat and stool samples, produced similar results. For his material, Buddingh chose cases of newborn diarrhea with stomatitis in older children which he considers a reflection of the same condition. In children over seven months of age, stomatitis is a more prominent sign rather than diarrhea. There is a sudden onset of anorexia and fine vesicular eruptions on the tip of the tongue which disappear in 24 hours. There is little temperature. The stomatitis lasts seven to fourteen days and is self-limiting. In those under six months there is a moderate diarrhea with greenish-yellow

stools flecked with blood. The occurrence of this condition in older children induces the occurrence of epidemic diarrhea in the newborn, according to this author. The question may legitimately be raised, however, as to whether this disease of which he speaks is truly similar to the epidemic form of diarrhea as reported most commonly. As previous mentioned, the classic symptomatology does not include bloody stools or transmission to older children. However, material[12] from one outbreak in Cincinnati was sent to him by Schwentker and included pharyngeal, stool and maternal vaginal samples. The filtrable agent was successfully isolated on rabbit corneas and identifiable by protection tests. Meikeljohn[29], using the same technique, was able to obtain positive results in 85 per cent of 256 cases from five hospitals. Further, he was able to pass the virus serially in rabbit corneas and chick embryos. In a few patients, neutralizing antibodies to the virus were found.

A second more promising method has been devised by Light and Hodes[31, 32] for isolating a possible causative agent. Using Seitz filtered saline suspensions from acutely ill infants, they were able to introduce this disease into calves. The material was obtained from three separate epidemics and passed to these animals both nasally and by stomach tubes. There followed a bloody, mucoid diarrhea after a two to five day incubation period. Successful passage of the virus has been attained in 84 calves to date. Symptoms included mild dehydration, anorexia and fever. The illness lasted eight to 54 days with a mortality of only 13 per cent. Immunity occurred in most of the survivors. Passage of the virus from the first to a second animal was also successful in two of three attempts. To eliminate the possibility of the agent being a member of normal flora, stools of healthy infants were similarly used but without effect. The activity of the virus remained after two months of freezing at minus 70° C., while boiling for ten minutes destroyed it. Protection tests with convalescent baby sera were successful, partially in some cases, completely in others.

Cummings[30] has also attempted the same method in chickens, mice, guinea pigs, rats and pigs but has been successful only with calves. In addition, the virus was passed serially through five animals. However promising Light and Hodes' method may be, a more simple and conclusive technique remains to be devised.

Unfortunately, no specific form of therapy has as yet been successful in combatting epidemic diarrhea. Penicillin[33, 34] and gamma

globulin have been entirely without effect. The result of sulfa therapy[35, 36] has been both favorable and unfavorable. Lyons[11] has used citrated blood of convalescent epidemic influenza victims to treat the infant diarrhea epidemic which resulted.

Supportive therapy[12] has been directed at combatting the dehydration and acidosis which develops so quickly. The intake of food is stopped until the diarrhea ceases. Saline infusions and soda bicarbonate are used to combat the dehydration. Parenteral fluids should include ascorbic acid, thiamine and amino acids to avoid protein depletion during the acute phase. Experience has proved that the only method of preventing progression of an epidemic is drastic action[4]. The entire obstetrical service and all the newborn nurseries should be closed immediately, cleaned, scrubbed and repainted. The sick should be isolated with their own utensils, treatment room and personnel, both nursing and medical. All exposures should likewise be segregated. Attempts at tracing the source of infection should be made with the aid and supervision of the public health department. Both mothers and personnel should be questioned for a history of illness, and appropriate cultures should be taken from all. The sanitation and nursing technique should be scrupulously investigated.

Since breaks in nursing technique are largely responsible for these epidemics, there must be rigid adherence to a program aimed at minimizing such risks. Ideally, a baby should have his own room or a minimum of 300 cubic feet[13]. Since this is usually impractical, a nursing unit of six to twelve newborns or six prematures[14], each with his own table and routine nursing supplies, should be the maximum number cared for by one nurse. The communal bathing and diapering tables and utility room should be eliminated[37]. Blanket nursing assignments should be abandoned if possible. Many subscibe to the idea of "rooming in[3, 4]" which, although more costly, reduces contacts and the exposure to communal equipment. At any rate, unnecessary trips to and from the mother as well as contacts with visitors, nurses and doctors should be avoided. There should be routine personnel examinations for upper respiratory infections and intestinal disturbances. The technique of formula sterilization should be rigidly enforced and routinely checked. It should also be unnecessary to mention the need for isolation and suspect nurseries. The former should especially be as far from the regular nursing facilities as pos-

sible[3, 14, 37]. Separate operating rooms should be used for regular and infected deliveries. The periodic cleansing of nurseries and delivery rooms is also a worthwhile consideration[3]. In those situations in which crowding is unavoidable, Chapple[38] has found the use of ultraviolet light and the evaporation of propylene glycol to be extremely useful in sterilizing the air. At least, under crowded conditions, attempts should be made to increase the rate of air circulation.

In conclusion, it may be said that infectious epidemic diarrhea of the newborn is in all probability caused by a miscellaneous etiological group bound together by symptoms of diarrhea. Assuming this, bacterial factors have actually been found in many cases by the discovery of contaminated nursing facilities. Attempts to incriminate a specific virus have so far proved equivocal, largely due to the lack of a simple specific test. The fact remains, carelessness of technique and supervision are the precipitating factors in fostering such epidemics. Prevention is, therefore, the best treatment. Sulfa and penicillin therapy have proved ineffective. With the advent of aureomycin and terramycin, encompassing a much broader spectrum, both bacterial and viral, a more hopeful outlook for the future should be entertained with respect to effective therapy.

BIBLIOGRAPHY

1. Rice, J.; Best, W.; Frant, S. and Abramson, H.: Epidemic Diarrhea of the Newborn. J.A.M.A., 109: 475, 1937.
2. Feemster, R.: Epidemiology of Infectious Diarrhea of the Newborn. Massachusetts Dept. Public Health, 1949.
3. Janeway, C.: Clinical Aspects of Infectious Diarrhea of the Newborn. Massachusetts Dept. Public Health, 1949.
4. Frant, S. and Abramson, H.: Epidemic Diarrhea of the Newborn. Brennemann's Practice of Pediatrics, Vol. 1, Chap. 28, Sec. ii, 1948.
5. Anderson, N. and Nelson, W.: Observations in Treatment of Epidemic Diarrhea. J. Pediat., 25: 319, 1944.
6. Feldman, F. and Anderson, J.: Epidemic Diarrhea—Review of the Literature. ARCH. PEDIAT., 64: 341, 1947.
7. High, R.; Anderson, N. and Nelson, W.: Further Observations of Epidemic Diarrhea. J. Pediat., 28: 407, 1946.
8. Foster, G.: Administrative Control of Epidemic Diarrhea. Massachusetts Dept. Public Health, 1949.
9. Hoag, L. and Kiser, W.: Acid-base Balance of the Newborn. Am. J. Dis. Child., 41: 1054, 1931.
10. Barenberg, L.; Greenstein, N.; Levy, W. and Rosenbluth, S.: Renal Thrombosis with Infarction Complicating Diarrhea of the Newborn. Am. J. Dis. Child., 62: 362, 1941.
11. Lyon, G. and Folsom, T.: Epidemic Diarrhea of the Newborn. Am. J. Child. Dis., 61: 427, 1941.
12. Swentker quoted by Dodd: Epidemic Diarrhea of the Newborn from the View of the Clinical Investigator. J. Pediat., 30: 700, 1947.
13. St. Gene, J.: Low CO_2 as a Prodrome of Infectious Diarrhea. J. Pediat., 22: 205, 1943.
14. Johnson, B. and Rothman, P.: Acidosis and Infectious Diarrhea. California & West. Med., 52: 229, 1940.
15. Clifford, S.: Diarrhea of the Newborn. New England J. Med., 237: 969, 1947.

16. McClure, W.: Role of Colon Organisms in Epidemic Diarrhea. J. Pediat., 22: 60, 1943.
17. Baker, C.: Epidemic Diarrhea—Three Outbreaks. J. Pediat., 14: 183, 1939.
18. Swentker, S.: Dysentery Due to B. dispar. Am. J. Child. Dis., 50: 1321, 1935.
19. Watt, J.: Etiology of Infectious Diarrhea. Am. J. Public Health, 35: 1205, 1945.
20. Barenberg, L.; Levy, W. and Grand, M.: Epidemiology of Infectious Diarrhea. J.A.M.A., 106: 1256, 1936.
21. Geiger, J. and Sappington, E.: Epidemic Diarrhea in San Francisco. ARCH. PEDIAT., 61: 134, 1944.
22. Rubenstein, A. and Foley, G.: Epidemic Diarrhea in Massachusetts—Ten Year Survey. New England J. Med., 236: 81, 1947.
23. Lembcke, P.; Quinlivan, J. and Orchard N.: Epidemic Diarrhea of the Newborn. Am. J. Public Health, 33: 1263, 1943.
24. Kneeland, Y.: Upper Respiratory Flora of Infants. J. Exper. Med., 51: 617, 1930.
25. Mayes, H.: Relation Between Bottle and Breast Feeding and Intestinal Flora. Am. J. Obst. & Gynes., 53: 285, 1947.
26. Gerstley, J.; Howell, K. and Nagel, B.: Factors Influencing Fecal Flora of Infants. Am. J. Child. Dis., 43: 555, 1932.
27. Campbell, K.: Report on Epidemic Diarrhea. M. J. Australia, 1: 79, 1945.
28. Buddingh, G.: Virus Stomatitis and Virus Diarrhea of Infants and Children. South M. J., 39: 382, 1946.
29. Meiklejohn, G.: Viral Studies on the Etiology of Epidemic Diarrhea of the Newborn. California & West. Med., 67: 238, 1947.
30. Cummings, G.: Epidemic Diarrhea from the View of an Epidemiologist and Bacteriologist. J. Pediat., 30: 706, 1947.
31. Light, J. and Hodes, H.: Isolation of Filtrable Agent Causing Diarrhea in Calves. Am. J. Public Health, 33: 1451, 1943.
32. Light, J. and Hodes, H.: Isolation of Filtrable Agent Causing Diarrhea in Calves. J. Exper. Med., 90: 113, 1949.
33. Allen, F.; Morrison, J. and Rutherford, W.: Penicillin Therapy in Infectious Diarrhea. Arch. Dis. Childhood, 21: 19, 1946.
34. High, R.; Pennsylvania M. J., 49: 1334, 1946.
35. Anderson, N. and Nelson, W.: J. Pediat., 1944.
36. Weymuller, C.; Beck, A. and Ittner, E.: Measures for the Protection of Newborn Infants. J.A.M.A., 133: 78, 1947.
37. Darrow, D.: J. Pediat., 28: 515, 1946.
38. Chapple, C.: Epidemic Diarrhea as a Hospital Problem. J. Pediat., 30: 710, 1947.

PREFRONTAL LEUKOTOMY IN MINORS. (Arquivos de Neuro-Psiquiatria, São Paulo, 7:349, Dec. 1949). Yahn and collaborators performed prefrontal leukotomy in 9 girls between the ages of 9 and 16 years with behavior disorders caused by epilepsy, oligophrenia and mental disease. Freeman and Watts' technic was used. The patients were inmates in a home for feebleminded girls. They were observed daily for more than one year after the operation. There was a recovery of normal personality in 1 patient and improvement in 4 patients with mental disease. The operation failed to improve 2. Two patients died from cerebral hemorrhage due to difficulties in placing the incision because of the abnormalities of the ventricles as previously shown by pneumoencephalography. The authors conclude that Freeman and Watts' leukotomy has a favorable effect on the conduct disorders in young persons. The degree of relief is greater than that achieved in chronic adult schizophrenics. The effect is due to a larger capacity for replacement possessed by immature brains, leading to a new neuropsychic orientation.—*Journal A.M.A.*

PEDIATRICS HALF A CENTURY AGO

From time to time the Archives, which was the first Children's Journal in the English language, will reprint contributions by the pioneers of the specialty over fifty years ago. It is believed that our readers will be interested in reviewing such early pediatric thought.

ABSCESS OF THE LUNG FOLLOWING ACUTE PNEUMONIA*

REPORT OF TWO CASES

L. EMMETT HOLT, M.D.
New York.

Abscesses of the lung of non-tuberculous origin are not uncommon in the autopsy room. They have been met with in my experience in about 7 per cent of the autopsies upon infants and young children dying from pneumonia. Such abscesses are generally small, varying in size from a pea to a small marble; occasionally one as large as a walnut is seen. On one occasion I met with one which occupied nearly the entire lobe of the lung. Abscesses seen postmortem are seldom single, and frequently the affected part of the lung is fairly honey-combed with abscesses, some of them being very minute and others larger. These are usually seen in the lungs of children of low vitality, or have followed very intense inflammation, sometimes a very prolonged one.

Abscess of the lung as a clinical condition in young children is rare, the cases here reported being the first that have come under my personal observation.

Huber has twice reported to this Society (Trans. Am. Pediat. Soc., Vol. III., p. 235; Vol. V., p. 206) cases of pulmonary abscess successfully treated by operation. One of these was in a child thirteen months old, the other in one four years old.

CASE I. ABSCESS IN THE UPPER LOBE OCCURRING TWO AND A HALF MONTHS AFTER ACUTE PNEUMONIA; RECOVERY AFTER ASPIRATION

J. P., male, eighteen months old, was admitted to the Babies' Hospital, December 30, 1902, with a history of having been taken

*Read before the Fifteenth Annual Meeting of the American Pediatric Society, held at Washington, D. C., May 12, 1903.
Reprinted from ARCHIVES OF PEDIATRICS, 21: 11-18, January 1904.

ill with fever and rapid breathing two months before; more or less fever since, but not much cough. For one week was much worse, high fever, prostration, etc. Having grown too sick to be treated at a dispensary he was referred to the hospital.

The family history was negative as regards tuberculosis, and no other facts could be gleaned from the personal history, as the parents were Italians and not very intelligent. On admission the child was found well developed and well nourished with no important signs of disease except in his chest.

The examination of the chest showed coarse râles over the right lung; over the left lung there was dullness both in front and behind, and over the upper portion in front the percussion note was almost flat; loud, coarse friction sounds were everywhere heard. The respiratory murmur was everywhere bronchial in character, but over the upper half of the lung very feeble in intensity. Although there was no evident displacement of the heart, the flatness was so marked that an exploratory puncture was made in two places. No fluid was found. The case was therefore regarded as one of pleuropneumonia and its termination in empyema was anticipated. The usual routine hospital treatment for pneumonia was adopted. The child continued to look very sick; the cough was distressing; the respiration always very rapid, and occasionally there were attacks of slight cyanosis.

For the first few days the temperature fluctuated irregularly between normal and 102.8° F. On the fifth day it took a sudden jump to 105° F. No marked changes in the physical signs occurred. The flatness over the upper lobe in front became gradually more and more pronounced, respiratory murmur was feeble, and friction sounds and coarse râles could be heard over nearly the whole chest.

On January 6, although the physical signs remained practically unchanged, the leukocytes had gone up from 24,200 to 45,000. The presence of pus was now regarded as practically certain, and exploratory puncture again made, this time in the third intercostal space just outside the mammary line; a syringe full of thick creamy odorless pus was withdrawn.

Still under the impression that we were dealing with a saccu-

lated empyema, preparations were made to remove the pus by incision. Chloroform was administered, but not to a degree to produce deep anesthesia. Desiring to drain the sac at the lowest possible point, a needle was inserted in the fourth intercostal space at the anterior axillary margin and pus found. An incision was then made at this point, but on opening the chest we came down upon the lung firmly bound by adhesions to the chest wall. The incision was extended upward and an opening made in the third intercostal space, but again the lung was met with as before.

The child bore the operation and the anesthetic badly; his color was so bad and pulse so weak that any further operative measures at this time seemed inadvisable. Examination of the pus drawn by puncture showed the pneumococci in numbers and a few staphylococci.

On the following day the chest was aspirated in the third interspace at a point midway between the axillary line and sternum and 1½ ounces of thick creamy pus were removed.

From this time there was steady improvement in the child's general condition although the temperature continued to fluctuate widely. On January 9 exploratory puncture was made in two places at the upper part of the left chest in front but no pus could be found. A further steady improvement in the physical signs together with a gradual fall in the leukocytes convinced us that no further accumulation of pus had taken place. Further exploration, therefore, was not made although the temperature continued without apparent reason until January 18. At this time the signs were dullness over the left upper lobe and feeble respiratory murmur; but the râles had gone; all the abnormal signs were much less distinct than at previous examinations and were gradually disappearing.

The boy remained in the hospital until March 15. During the last month he gained three pounds in weight. The respiration continued somewhat rapid and he had a moderate cough evidently from the extensive pleuritic adhesions. On looking up the case subsequently it was learned that he had remained well for about a month after his discharge from the hospital, when he was taken severely ill with diphtheria and died in twenty-four hours.

CASE II. ABSCESS IN THE LEFT LOWER LOBE TEN WEEKS AFTER ACUTE LOBAR PNEUMONIA. RECOVERY AFTER INCISION AND DRAINAGE

H. P., male, two years, ten months; admitted to hospital, January 26, 1903, with signs pointing to a lobar pneumonia of the left lower lobe. The symptoms and physical signs were fairly typical. He had been acutely ill for five days. The family history was negative as far as tuberculosis or any hereditary disease was concerned. The patient had had bronchitis when an infant of six months, but had not been otherwise ill up to the commencement of the present attack. His temperature on admission was 104° F., pulse 140, respiration 56, leukocytes 39,500.

By February 1, the temperature had fallen gradually to normal, but remained so only a single day. The bronchial breathing disappeared but the dullness remained; and as the leukocyte count ranged from 35,000 to 50,000, it was expected that empyema would develop. The child was pale, had a poor appetite, looked badly, and lost weight. A slight, irregular temperature continued for the next five weeks. The physical signs remained practically unchanged; at no time was there marked flatness but always moderate dullness over the left lower lobe, together with feeble bronchovesicular breathing, pleuritic friction sounds and coarse râles. We concluded it was probably a delayed resolution. On March 9, he was removed from the hospital by his mother. The last leukocyte count on February 24 showed 21,700.

On March 23, at the end of two weeks, he was re-admitted, having had in the interval an acute otitis which had ruptured, and the ear was discharging pus. From this time there was more temperature, prostration, and general evidence of illness. Examination of the chest showed no very great change in the physical signs. There was still present moderate dullness over the left lower lobe behind, with diminished voice and feeble breathing of a bronchovesicular quality; a few friction sounds were heard; leukocytes 28,000. We felt tolerably sure now that empyema was developing, but greatly to our surprise the temperature soon fell to normal and remained there for four days. In spite of this there was no improvement in the symptoms and by March 30, a rapid increase of the leukocytes to 63,000 convinced us that pus must be present. An

exploratory puncture was made near the angle of the scapula with negative results. A second puncture at the same level, but a little nearer the spine, brought a syringe full of thick yellow pus. The examination of this showing only the pneumococcus, a diagnosis of sacculated empyema was made without hesitation.

On March 31, the patient was given chloroform and further punctures made. It was not, however, until the fourth aspiration that pus was finally obtained, this being about one space higher than the site of the successful puncture previously. An incision was made at this point but we came down upon the lung firmly bound to the chest wall by old adhesions. A needle was inserted into the lung through the incision and pus found at a depth of about 1½ inches; there seemed now no possible doubt that we were dealing with an abscess of the lung. The firm fibrous adhesions which bound the lung to the chest wall obliterated the pleural cavity at this point, and made further operation comparatively easy. Following cautiously the line of the exploring needle an opening was slowly bored into the lung with a closed artery clamp, and a cavity reached at about the depth indicated, from which a gush of pus took place. About two ounces of thick creamy odorless pus was evacuated. The opening was enlarged with the finger and a rubber drainage tube inserted and the usual dressings of empyema applied.

The progress of the case from this time was uneventful. The discharge was quite abundant for four or five days; the drainage tube was gradually shortened, and after three weeks permanently removed. In another week the wound had healed completely.

The improvement in the child's general condition after the operation was immediate and striking. The cough disappeared, the boy gained rapidly in weight, and he was finally discharged from the hospital on May 25; the only remaining signs being slight dullness and rather feeble breath sounds of a quality nearly normal. This child was heard from about four months after his discharge and reported to be well.

Remarks. Both of these cases followed acute pneumonia, the abscess being recognized about two and a half months after the attack of primary pneumonia. In Case II. the child was under observation almost continuously. In Case I., the history of the onset was fairly definite, and there can be little doubt as to the

time when the process in the lung began. It is probable that a small abscess had been slowly forming for several weeks previous to its being recognized, and that it occurred in the center of an inflammatory area which had not resolved.

The bacteriological examinaions of the pus are interesting. In both the pneumococcus was present; in one case it was associated with the staphylococcus; in neither case were tubercle bacilli found. These findings indicate that the abscess had been shut off from the bronchi by the products of inflammation, otherwise we would have had more evidence of mixed infection and doubtless foul pus. The presence of the pneumococcus gave additional weight to the supposition that we were dealing with an empyema. The fact that no pus was coughed up either before or after the operation also shows that no communication with the bronchus existed, although if the case had been allowed to go on it is probable that such would have been the result.

Perhaps the chief interest of cases like these is in the diagnosis. The literature of the subject shows that most of the cases of pulmonary abscess have been operated upon with the idea that a sacculated empyema was present, and indeed that was the first diagnosis made in both of my cases. In reviewing these histories critically, there are, however, a few points of differentiation which I believe may be of value in subsequent cases.

Percussion gives no clue to the nature of the disease. In both pulmonary abscess and sacculated empyema we have a more or less general dullness over the lobe, with flatness over a somewhat limited area. Again the voice and breathing sounds are practically identical in the two conditions. In both of my cases the presence of loud, coarse pleuritic friction sounds accompanying the signs on percussion and auscultation just mentioned, are I believed of much importance. Such signs could hardly exist if the accumulation of pus was in the pleural cavity. Another point of some value is the difficulty of finding the pus with the exploring needle, or finding it at one time and later not finding it even when a puncture was made in the same intercostal space. This should be regarded as evidence that the pus is deep-seated; not only must the needle be inserted in the same space but it must follow the same direction in order to find the pus. An abscess the size of a hen's egg, situated somewhat deeply in the lung might easily be missed although a

half dozen punctures were made into the lobe which contained it.

A slight displacement of the heart would be probable in nearly all cases of empyema unless the amount of pus is very small. Of course, it would not be present in an abscess unless the amount of pus were large. This, therefore, could not be depended upon as an absolute point of differentiation.

A pulmonary abscess and a persistent or unresolved pneumonia may easily be confounded by physical signs. If an abscess exists it is often in the center of such a pneumonic area. Percussion gives little definite information; dullness often marked is present in both conditions. The circumscribed area of flatness would be found only when the abscess was large or superficially situated. Breathing and voice sounds over an abscess are feeble or absent, while over an unresolved pneumonia they are usually bronchial in character, though sometimes feeble in intensity. Coarse friction sounds are likely to be found in both conditions, often over the entire lobe if it were the seat of the antecedent acute pneumonia.

The physical signs of abscess, therefore, are not very characteristic. Additional help may be obtained from the other symptoms, especially the presence of continued fever and a high leukocytosis. The temperature curve in abscess is not characteristic, but some continuous fever is almost certain to be present. Fever is more constant and altogether higher than is usual in an unresolved pneumonia. A leukocytosis of 15,000 to 25,000 is often present in unresolved pneumonia, but a count of 40,000 or 50,000 and especially a rapid increase in the leukocytosis points strongly to the presence of pus.

From tuberculosis the diagnosis of abscess may be difficult and in some cases impossible without a prolonged period of observation. It should be remembered that abscess most frequently follows an attack of acute lobar pneumonia, that the patient may have been previously in the best of health and without any exposure to tuberculosis. Wasting is a less frequent symptom, tubercle bacilli are not found in the sputum and the pus found by puncture contains pneumococci, but no tubercle bacilli.

The treatment in the cases reported was comparatively simple. It was expected that the abscess would refill in the first case after aspiration. Incision is certainly to be preferred as a general method of treatment, provided the child's general condition is such as to

allow the operation. Inamuch as the pleural cavity is generally obliterated by dense adhesions, as in both these cases, the opening in the lung can be made with very little difficulty or risk.

ROLE OF ADRENAL IN PINK DISEASE. (Medical Journal of Australia, Sydney, 1: 363, March 10, 1951). Williams and co-workers found no constant or significant disturbance of the serum sodium content of 22 patients with pink disease (acrodynia) as compared with 22 normal children of the same age, although two patients had a low serum sodium level. The hematocrit, hemoglobin, and protein values were raised, and the serum chloride level was lowered in pink disease, but the mechanism of these changes was not clear. There was no evidence of hypofunction of the adrenal gland as determined by studies of blood sodium and potassium levels and 11-oxysteroid excretion in the urine. A controlled clinical experiment on 10 selected patients failed to show any really significant difference in the recovery rate of the five treated with salt and desoxycorticosterone as compared with those who did not receive this treatment.—*Journal A.M.A.*

TUBERCULOUS MENINGITIS CURED BY STREPTOMYCIN. (Archivos de Pediatría del Uruguay, Montevideo, 22:18, Jan. 1951). A boy, 10, in the course of three years after occurrence of primary tuberculous infection successively had tuberculous pleurisy, ascites, and bilateral spread of pulmonary tuberculosis, which were controlled by the administration of dihydrostreptomycin, in a daily dose of 1 gm. up to a total dose of 40 gm. Four months later, the tuberculous meningitis developed. The diagnosis was confirmed by the positive results of cultures and inoculation of the cererospinal fluid in animals. The disease was cured by dihydrostreptomycin, given intramuscularly in a dose of 2 gm. daily and, later on, of 25 mg. three times a week, up to a total dose of 372 gm. in six months. The intraspinal dose was 50 mg. at four day intervals, up to a total dose of 1.80 gm. for the first few weeks of the disease. The drug was well tolerated by the intramuscular route but not by the intraspinal route. Cure has been maintained up to the present (four months after treatment).—*Journal A.M.A.*

DEPARTMENT OF ABSTRACTS

LANS, H. S.; STEIN, I. F.; BECKER, R. J.; HOYNE, A. L. AND MEYER, K. A.: POTASSIUM DEFICIENCY IN BULBAR POLIOMYELITIS. (Journal American Medical Association, 146:1017, July 14, 1951).

Potassium deficiency is being recognized in many clinical conditions, including infantile diarrhea, alkalosis due to intestinal and pyloric obstructions and vomiting and in many postoperative patients. The authors report on three cases of bulbar poliomyelitis (one in a woman of 29, another in a woman of 18 and a boy of 9) in which studies of electrolyte metabolism showed potassium deficiency. Certain signs and symptoms are associated with potassium deficiency, such as loss of strength and energy, listlessness and anorexia. Weakness of the extremities develops and progresses to flaccid paralysis. Cranial nerves are rarely involved. Weakness of respiratory muscles is often marked; paralysis of the intercostal muscles and diaphragm may be present. Breathing becomes rapid and shallow. In the absence of prompt potassium therapy, respiratory paralysis and death may ensue. Patients with bulbar poliomyelitis cannot tolerate the superimposed symptoms of potassium deficiency. When potassium deficiency is found to be present, 2 to 3 gm. of potassium chloride should be added to each liter of intravenous fluid so that the patient receives 6 to 12 gm. of potassium chloride a day. MICHAEL A. BRESCIA, M.D.

THWAITE, W. G.: BRONCHIOGENIC CARCINOMA IN A TWELVE-YEAR-OLD BOY. (Journal of the Medical Association of Georgia, 40:216, May 1951).

The author reports a case of bronchiogenic carcinoma in a 12-year-old negro boy. He complained of pain in the left chest of one week's duration in addition to a nonproductive cough. The boy appeared ill, poorly nourished and had increased respirations. The chest showed impaired percussion note beneath the left scapula extending to the left infra-axillary area; there were crepitant râles over this same area. Eight days later the cough was said to be better but the chest pain was worse and involved the left anterior area as well. Patient was admitted to the hospital at which time x-ray of the chest showed marked density of most of the left chest

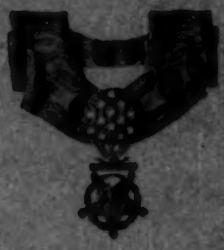
with the heart displaced to the right. A chest tap was done and about 300 cc. of blood-tinged fluid was obtained. The boy rapidly went down hill and died in less than two months. At autopsy the left pulmonary pleura was grey in color and adherent to the displaced pericardium and to the parietal pleura. It had the feel of a thickened crust or shell containing fluid. On cutting through this this shell of pleura and lung periphery, it was a shell indeed filled with grey lung tissue reduced to a liquid and soft solid state.

MICHAEL A. BRESCIA, M.D.

SCHACHTER, M. AND COTTE, S.: A STUDY ON THE IMPORTANCE OF THE SEX FACTOR IN EARLY DEVELOPMENT. (Il Lattante, 22:144, March 1951).

In comparing the dentition of 522 male infants with 310 female infants, it was found that 3.6 per cent of the males and 6.4 per cent of the females had early dentition and 84.8 per cent of the males and 75.4 per cent of the females had normal dentition while 11.5 per cent of the males and 18 per cent of the females had delayed dentition. In comparing the onset of walking in 612 males and 369 females, it was found that 41.5 per cent of the males and 41.7 per cent of the females walked early, and normal walking was found in 55.7 per cent of the males and 44.4 per cent of the females, while late walking was found in 2.7 per cent of the males as compared to 13.8 per cent in the females. In comparing the onset of the use of words in 311 males and 164 females, early use of words was found in 43 per cent of males and 58 per cent of females, normal speech in 46.9 per cent of the males and 35.9 per cent in the females, while late speech was found in 9.9 per cent of the males as compared to 5.5 per cent of the females. In comparing 205 males and 88 females as to time of controlling bed wetting, they found 6.3 per cent of the males and 11.3 per cent of the females were dry at the end of 12 months, dryness occurring between the 13th and 24th month occurred in 22.9 per cent of the males and 21.5 per cent of the females, between the 25th and 36th month in 18.5 per cent of the males and 25 per cent of the females and control developing after 36 months in 52.2 per cent of the males and 42 per cent of the females. MICHAEL A. BRESCIA, M.D.

Medal of Honor



Lieutenant Frederick Henry of Clinton, Oklahoma—Medal of Honor for sacrificing himself to save his platoon in combat near Am-Dong, Korea, September 1, 1950. When the platoon could no longer hold its position, Lieutenant Henry ordered the men to pull back. But someone had to stay behind to provide covering fire. He chose to be that man, and was lost.

Always remember this—Lieutenant Henry offered his life for more than just a small platoon in far-away Korea. It was also for America. For you.

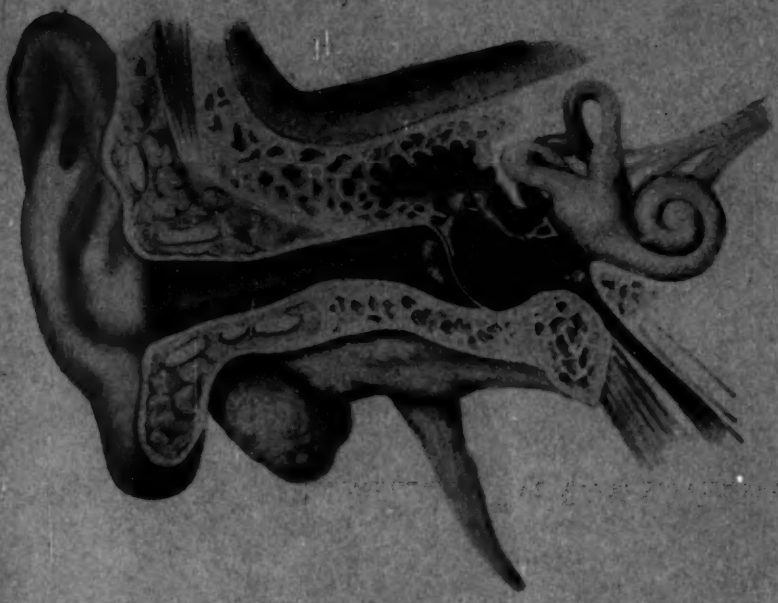
Isn't there something you can do when this man did so much? Yes, there is. You can help keep the land he loved solid and strong and secure. You can do a job for defense . . . by buying United States Defense* Bonds, *now!* For your bonds give your country greater strength. And a strong America is your best hope for peace and freedom—just as it was his.

Defense is your job, too. For the sake of *every* man in service, and for yours, start buying more United States Defense Bonds now.

Remember when you're buying bonds for defense, you're also building personal cash savings. Remember, too, if you don't save *regularly*, you generally don't save at all. So sign up in the Payroll Savings Plan where you work, or the Bond-A-Month Plan where you bank. For your country's security, and your own, buy United States Defense Bonds!

U.S. Savings Bonds are Defense Bonds - Buy them regularly!

The U. S. Government does not sign for this advertisement. It is donated by this publication in cooperation with the Advertising Council and the American Publishers of America as a public service.



in upper respiratory infections:

"Our cases of bronchopneumonia, otitis media, tonsillitis, sinusitis, and laryngotracheo-bronchitis responded . . . rapidly to terramycin."

Pottenfield, T. C., and Stanbrough, C. A.: J. Philadelphia General Hosp. 2:0 (Jan.) 1951.

CRYSTALLINE TERRAMYCIN HYDROCHLORIDE

available | *Capsules, Elixir, Oral Drops, Intravenous, Ophthalmic Ointment, Ophthalmic Solution.*

ANTIBIOTIC DIVISION **Pfizer** CHAS. PFIZER & CO., INC., *Brooklyn 6, N. Y.*